

Northumbria Research Link

Citation: Cornelissen, Katri (2016) What does it mean to have distorted body image in anorexia nervosa? Doctoral thesis, Northumbria University.

This version was downloaded from Northumbria Research Link:
<https://nrl.northumbria.ac.uk/id/eprint/30330/>

Northumbria University has developed Northumbria Research Link (NRL) to enable users to access the University's research output. Copyright © and moral rights for items on NRL are retained by the individual author(s) and/or other copyright owners. Single copies of full items can be reproduced, displayed or performed, and given to third parties in any format or medium for personal research or study, educational, or not-for-profit purposes without prior permission or charge, provided the authors, title and full bibliographic details are given, as well as a hyperlink and/or URL to the original metadata page. The content must not be changed in any way. Full items must not be sold commercially in any format or medium without formal permission of the copyright holder. The full policy is available online: <http://nrl.northumbria.ac.uk/policies.html>



**Northumbria
University**
NEWCASTLE



UniversityLibrary

Northumbria Research Link

Citation: Cornelissen, Katri (2016) What does it mean to have distorted body image in anorexia nervosa? Doctoral thesis, Northumbria University.

This version was downloaded from Northumbria Research Link:
<http://nrl.northumbria.ac.uk/id/eprint/30330/>

Northumbria University has developed Northumbria Research Link (NRL) to enable users to access the University's research output. Copyright © and moral rights for items on NRL are retained by the individual author(s) and/or other copyright owners. Single copies of full items can be reproduced, displayed or performed, and given to third parties in any format or medium for personal research or study, educational, or not-for-profit purposes without prior permission or charge, provided the authors, title and full bibliographic details are given, as well as a hyperlink and/or URL to the original metadata page. The content must not be changed in any way. Full items must not be sold commercially in any format or medium without formal permission of the copyright holder. The full policy is available online: <http://nrl.northumbria.ac.uk/policies.html>



**Northumbria
University**
NEWCASTLE



UniversityLibrary

WHAT DOES IT MEAN TO HAVE DISTORTED BODY IMAGE IN ANOREXIA NERVOSA?

K K CORNELISSEN

PhD

2016

WHAT DOES IT MEAN TO HAVE DISTORTED BODY IMAGE IN ANOREXIA NERVOSA?

KATRI K CORNELISSEN

A thesis submitted in partial fulfilment of the
requirements of the University of Northumbria at
Newcastle for the degree of Doctor of Philosophy

Faculty of Health and Life Sciences

June 2016

Abstract

This thesis explores body image distortion in anorexia nervosa spectrum disorders (ANSD), using 3D computer generated (CGI) stimuli together with psychophysical and behavioural methods. Experiments 1-3 on healthy controls show that not only does participants' psychological state influence the perception of their body size, but so does their actual body size, indexed by Body Mass Index (BMI). Healthy controls whose BMI is closest to the population average estimate body size accurately, those with lower than average BMIs over-estimate and those with higher than average BMIs under-estimate, demonstrating contraction bias for body size estimations. Furthermore, control participants' sensitivity to changes in body size follows Weber's law: larger bodies require proportionately bigger differences between them to be discriminated. In marked contrast, ANSD individuals with the lowest BMI are the most accurate and most sensitive at body size estimation. As these participants' BMI increases, sensitivity reduces dramatically, and participants rapidly start to overestimate their body size. Experiment 4 mapped the relationship between participants' behavioural judgements of body size and where they look during the task. This study showed that (i) accurate body size estimators tended to look more in the waist region, and this was independent of clinical diagnosis; (ii) there is a pattern of looking at images of bodies, particularly viewing the upper parts of the torso, which is specific to participants with ANSD but which is independent of accuracy in body size estimation. Experiment 5 is an adaptation based training program for ANSD participants. It succeeded in raising individuals' perceptual boundary for thin versus fat body sizes. This resulted in reduced psychological concerns about ANSD participants' own body shape. In conclusion, this thesis brings novel insight into the phenomenon of distorted body image in ANSD. Implications for the treatment of eating disorders are discussed based on these new data.

Table of Contents

Abstract	2
List of Tables	6
List of Figures	7
Publications and submitted materials	9
Acknowledgements	10
Declaration	11
Chapter 1: Introduction	12
1.1. Diagnostic criteria	13
1.2. Difficulties with diagnostic criteria	17
1.3. Aetiology of the eating disorders	21
1.3.1. Genetic influences	22
1.3.2. Psychological influences	27
1.3.3. Environmental influences	28
1.3.4. Body Image Distortion (BID), an aetiological perspective	31
1.4. Neural correlates of Eating disorders	34
1.5. Body Image Distortion (BID), definition, mechanism and measurement	38
1.5.1. Components of BID	39
1.5.2. Measuring the attitudinal aspects of BID	40
1.5.2.1. Body dissatisfaction	41
1.5.2.2. Self-esteem, mood and depression	43
1.5.3. Measuring the perceptual aspects of BID	44
1.6. Measuring actual body size	48
1.7. Comparing BID in eating disordered participants and controls	51
1.8. Contraction bias and the role of body mass index (BMI)	56
1.9. Non-body size estimations in eating disordered participants and controls	59
1.10. Treatment methods for eating disorders	60
1.10.1. Specific therapies for Body Image Distortion (BID)	62
1.11. Research questions	63
Chapter 2: Methods	65
2.1. Computer Generated Imagery (CGI) for stimulus creation	65
2.1.1. Calibrating model BMI	67
2.2. Classical psychophysical methods	71
2.2.1. Method of constant stimuli	72
2.2.2. Method of limits	74
2.3. Psychometric measures	75
Chapter 3: Experiments 1 and 2	78
3.1. Introduction	78
3.2. Experiment 1: Contraction bias	79
3.3. Methods	79
3.3.1. Participants	79
3.3.2. Stimuli	80
3.3.3. Procedure	80
3.4. Results	81
3.5. Experiment 2: Just Noticeable Difference (JND)	83
3.6. Methods	83

3.6.1. Participants	83
3.6.2. Stimuli	83
3.6.3. Procedure	84
3.7. Results	86
3.8. Discussion	87
Chapter 4: Experiment 3	90
4.1. Introduction	90
4.2. Methods	93
4.2.1. Participants	93
4.2.2. Anthropometric measurements	94
4.2.3. Psychometric measurements and procedure	94
4.2.4. Psychophysical measurements and procedure	94
4.3. Results	94
4.3.1. Reliability measures	94
4.3.2. ANSD participant characteristics	95
4.3.3. Comparison between ANSD and control participants	99
4.3.3.1. PSE	100
4.3.3.2. DL	103
4.4. Discussion	103
Chapter 5: Experiment 4	110
5.1. Introduction	110
5.2. Methods	113
5.2.1. Participants	113
5.2.2. Psychometric and anthropometric measurements	114
5.2.3. Stimulus image preparation	115
5.2.4. Psychophysical measurement procedures	116
5.3. Results	117
5.3.1. Psychometric task reliability	117
5.3.2. Univariate statistics	117
5.3.3. Whole sample analysis: psychometric measures	118
5.3.4. Whole sample analysis: body size over-estimation	119
5.3.5. Whole sample analysis: comparisons between groups	120
5.4. Discussion	125
Chapter 6: Experiments 5 and 6	132
6.1. Introduction	133
6.2. Methods	135
6.2.1. Participants	135
6.2.2. Psychometric and anthropometric measurements	136
6.2.3. Stimulus image selection	136
6.2.4. Perceptual training paradigm	137
6.2.5. Procedure	139
6.3. Results	139
6.4. Discussion	143
6.5. Experiment 6: Rationale	144
6.6. Methods	144
6.6.1. Participants	144
6.6.2. Psychometric and anthropometric measurements	145

6.6.3. Procedures.....	146
6.7. Results	146
6.8. Discussion	149
Chapter 7: Experiment 7	153
7.1. Introduction	153
7.2. Methods	156
7.2.1. Participants	156
7.2.2. Psychophysical measurements.....	158
7.2.3. Eye movement recordings	159
7.2.4. Eye movement analysis path	160
7.3. Results	163
7.3.1. Univariate statistics.....	163
7.3.2. Multivariate statistics.....	164
7.3.2.1. Behavioural responses in psychophysical tasks.....	164
7.3.2.2. Comparisons of fixation density between groups.....	165
7.3.2.3. Fixation density as a function of stimulus BMI.....	167
7.4. Discussion	170
7.4.1. The role of the face	170
7.4.2. Potential confounds in group comparisons.....	171
7.4.3. Perceptual versus cognitive factors	173
7.4.4. Limitations.....	174
7.4.5. Treatment implications	175
Chapter 8: Summary and General Discussion	176
8.1. Overview	176
8.2. Research aims.....	176
8.3. Summary of research findings.....	177
8.3.1. Chapter 3.....	177
8.3.2. Chapter 4.....	177
8.3.3. Chapter 5.....	178
8.3.4. Chapter 6.....	179
8.3.5. Chapter 7.....	179
8.4. General discussion.....	180
8.4.1. Clinical implications.....	181
8.4.2. Limitations.....	182
8.4.3. Future research: the importance of body ownership.....	185
8.5. Conclusions	187
References	188

List of Tables

Table 4.1: Characteristics of the three ANSD subgroups in Experiment 3	96
Table 4.2: Means and standard deviations for the participant characteristics, separated according to whether they belong to the ANSD or the healthy control group in Experiment 3	98
Table 5.1: Characteristics of the participants in Experiment 4	114
Table 5.2: Pearson correlations between psychometric measures from the whole sample in Experiment 4	118
Table 5.3: Pearson correlations between PSYCH, chronological age and body size over-estimation across all participants in Experiment 4.....	120
Table 5.4: Pairwise comparisons of AVERAGE and IDEAL BMI estimates between the ANC, ANH and CON groups of participants, computed for actual BMIs of 15, 25 and 30.	123
Table 5.5: Pattern of differences between the AVERAGE and IDEAL body size estimates and actual BMI in Experiment 4. The p-values represent tests of location against the null hypothesis: $H_0 : \mu_0 = 0$	125
Table 6.1: Summary table of the demographic and questionnaire data from the participants in Experiment 5.....	135
Table 6.2: Summary table of the psychological outcomes from Experiment 5	142
Table 6.3: Summary table of the demographic and questionnaire data from the participants in Experiment 6.....	145
Table 6.4: Summary table of the psychological outcomes from Experiment 6.	149
Table 7.1: Participant characteristics for Experiment 7	158
Table 7.2: Stimulus images contributing to three BMI sub-groups	168

List of Figures

Figure 1.1: Overview of energy homeostasis in rat	24
Figure 1.2: Left hemisphere MR sagittal slices, at three MNI x-coordinates, showing the main sites for differences between patients with AN and healthy controls from structural and functional MR studies, as summarised in Gaudio and Quattrocchi (2012).....	36
Figure 1.3: Tripartite model of the emergence of bulimia nervosa from van den Berg et al. (2002).	41
Figure 1.4: A) The top row shows three of our CGI stimuli representing BMI 13, 17 and 21 respectively. The second row shows the body shape changes produced by applying the video distortion technique (VDT) to the central image in the first row.	47
Figure 1.5: Sketch graphs to illustrate the single and dual channel models	57
Figure 2.1: Scatter plots of BMI as a function of the hip and waist circumferences, height and age of the 4976 Caucasian females in the HSE 2008 dataset.	67
Figure 2.2: A plot of CGI model waist and hip circumference as a function of animation frame number, together with the location of the animation Key Frames (see text)	70
Figure 2.3: Representative 3D rendered stimuli at three different BMI levels.....	71
Figure 2.4: A graphical illustration of how the psychometric function for body size estimation can be used to separate out pure sensory sensitivity (indexed by the difference limen, DL) from perceptual bias (indexed by the point of subjective equality, PSE).	72
Figure 2.5: An illustration of the body shape changes for an anonymised, bespoke avatar in Experiment 4, as the slider control is moved from left to right through screenshots A, B, C & D.....	75
Figure 3.1: Scatterplot depicting the relationship between the actual weight of the women in the images (kg) and the mean of the participants' estimations of their weight (i.e. one data point represents one image).	81
Figure 3.2: A) is a plot of mean JND as a function of the reference BMI value for each of the BMI ranges for the two CGI 3D models. The two models are indicated by the upward and downward pointing cyan triangles, respectively. Error bars represent 1 s.e. of the mean.....	86
Figure 4.1: A) Schematic representation of the results from Cornelissen et al. (2013) in which women with AN and controls used an interactive software program to estimate body size.....	91

Figure 4.2: (A) Shows the relationship between participants' BMI (x-axis) and their subjective estimate of body size (PSE) with the effects of PSYCH statistically controlled.	101
Figure 5.1: Illustration of real body shape variation, across each row, in individuals with approximately the same BMI.	111
Figure 5.2: Plots of estimated BMI as a function of actual BMI for the data averaged across the belief, leg and torso conditions in A and the ideal condition in B. Red, pink and blue dots with their respective regression lines represent: ANC, ANH and CON participants.	122
Figure 5.3: Illustration of the proposal that different body size distributions are used by observers as the 'reference' for the 2AFC task (Population at large) and the method of limits task (Experience of self).....	128
Figure 6.1: The middle row shows part of the body sequence varying in BMI from lower to higher. The top row illustrates the results from a baseline assessment and the position of the categorical boundary prior to training.	137
Figure 6.2: A) A plot of the mean value of BMI at the categorical boundary, predicted from the multi-level model as a function of measurement day. Magenta and red circles represent control group pre- and post-training thresholds respectively.	140
Figure 6.3: A) A plot of the mean value of BMI at the categorical boundary, predicted from the multi-level model as a function of measurement day. Cyan and blue circles represent pre- and post-training thresholds respectively for the ANSD participants in experiment 2.	147
Figure 7.1: Maps of the relationship between psychophysical performance in body size estimation and where observers look on the body.	166
Figure 7.2: Fixation density as a function of stimulus image BMI grouping	169
Figure 8.1: Representations of body shape change in a female, aged 25, height 1.6m, as a function of BM, courtesy of Perceiving Systems, MPI, Tübingen.	183

Publications and submitted materials

Cornelissen KK, McCarty K, Bester A, Cornelissen PL & Tovée MJ (2016) Measuring body image distortion in anorexia nervosa with 3D avatars. (in preparation). *A, B, C, D*

Cornelissen KK, Seed, J & Manning, J (2016) 2D:4D in eating disorders. (in preparation). *A, B, C*

Gledhill LJ, Cornelissen KK, Cornelissen PL & Tovée MJ (2016) An interactive training program to treat body image disturbance. (submitted). *A, B, C, D*

Cornelissen KK, Cornelissen PL, Hancock PJB & Tovée MJ (2016) Fixation patterns, not clinical diagnosis, predict body size over-estimation in eating disordered women and healthy controls. *International Journal of Eating Disorders* (in press). *A, B, C, D*

Cornelissen KK, Gledhill LJ, Cornelissen PL & Tovée MJ (2016) Visual biases in judging body weight. *British Journal of Health Psychology*. DOI: 10.1111/bjhp.12185. *A, B, C, D*

Cornelissen KK, Bester A, Cairns P, Tovée MJ and Cornelissen PL (2015) The influence of personal BMI on body size estimations and sensitivity to body size change in anorexia spectrum disorders. *Body Image*, 13, 75-85. *A, B, C, D*

A = major contribution to writing

B = major contribution to design

C = major contribution to data collection

D = major contribution to data analysis

Acknowledgements

First, I would like to thank Prof. Piers Cornelissen for his support both personally and at work, and for his guidance and insistence over the past three years. Despite many arguments, upsets, disappointments, the conversations we have had, the last three years have been extremely fruitful and as a result I am very happy how I have grown as a researcher. Secondly, I would like to thank Dr. Martin Tovée for his invaluable support, and ability, when needed, to bring things back down to earth. I thank Dr. Nick Neave for his guidance with ethics applications and willingness to read and comment on my paper drafts.

For support and multiple gym visits to take me out of the office, many thanks to my colleagues from the Cognition and Communication Laboratory, especially: Richard Cutter, Sarah Allen, Mia Campbell, Ash Chapman, Kamila Irvine, Jo Greer and Kris McCarty.

None of this would have been possible without the support of my parents, Helena and Tapio. They have always backed me, struggled with me through my own eating disorder, and helped me to have belief in myself. Thank you for the numerous conversations we have had, and for listening during those tough times. Thank you also to my brother Vesa for inspiring me, distracting me and helping me to keep my feet on the ground.

I dedicate this thesis to my two children, Agi and Isak. These two boys have kept me going forward in both my personal and working life. Their pictures blue tacked to the side of my computer screen gave the extra strength to keep going. I wouldn't have coped with all the stress and disappointment on the way if I had not had those two faces to amuse, comfort and challenge me outside work.

Declaration

I declare that the work contained in this thesis has not been submitted for any other award and that it is all my own work. I also confirm that this work fully acknowledges opinions, ideas and contributions from the work of others.

Any ethical clearance for the research presented in this thesis has been approved. Approval has been sought and granted by the Faculty of Health and Life Sciences Ethics Committee.

I declare that the Word Count of this Thesis is 46,381.

Name: Katri Cornelissen

Signature:

Date: 7th June 2016

Chapter 1: Introduction

Eating disorders are not a new phenomenon. Roman de la Rose transcripts from the medieval period contain descriptions of deliberate self-starvation, unrelated to religious beliefs. The concept of disordered eating as a medical problem was realized in 1689 by Richard Morton who reported an individual who was starved not due to their life circumstances, and described the sufferer as having “a nervous consumption” and who appeared as a "skeleton only clad with skin". The term anorexia nervosa (AN) (literally "nervous absence of appetite") was introduced by William Gull (1873). For nearly a century thereafter, disordered eating was considered strictly as a medical disease, and the treatment was based on regular feeding and supervision of the feeding behaviour. However, by 1960 (see e.g., Bliss & Branch, 1960) clinicians began to realize that anorexia nervosa had a psychological dimension beyond the evidence of dramatic weight loss, thereby setting the scene for Hilde Bruch (Bruch, 1962) to introduce a specific set of emotional disturbances related to AN: disturbance of body image, disturbance of perception and an overwhelming sense of ineffectiveness. Since that time, anorexia nervosa has primarily belonged in the domain of psychiatry. Moreover, the list of eating disorders has expanded. For example, bulimia nervosa was first described as late as 1979 (Russell, 1979). It is an open question whether this is due to a belated recognition of the disorder, or whether bulimia nervosa may have emerged, like obesity in Western society, from an environment in which cheap, highly calorific food has become readily available, and cultural norms reward thinner bodies. Currently, as outlined in the Diagnostic and Statistical Manual 5 (DSM-5; American Psychiatric Association, 2013), there are 4 main categories of eating disorder as follows.

1.1. Diagnostic criteria

1) **Anorexia Nervosa.** The DSM-5 criteria include:

- A. Persistent restriction of energy intake relative to requirements leading to a significantly low body weight in the context of age, sex, developmental trajectory, and physical health. Significantly low weight is defined as a weight that is less than minimally normal, or, for children and adolescents, less than that minimally expected.
- B. Intense fear of gaining weight or becoming fat, or persistent behaviour that interferes with weight gain, even though at a significantly low weight.
- C. Disturbance in the way in which one's body weight or shape is experienced, undue influence of body weight or shape on self-evaluation, or persistent lack of recognition of the seriousness of the current low body weight.

DSM-5 recognises two different subtypes: Restricting Type in which during the last three months, the person has not engaged in recurrent episodes of binge eating or purging behaviour (i.e., self-induced vomiting or the misuse of laxatives, diuretics, or enemas), and bingeing-eating/purging type in which the person *has* engaged in these behaviours over the last three months.

The criteria for anorexia nervosa have changed from the DSM-IV, particularly with regard to criterion A, in order to facilitate diagnosis. Criterion A in the DSM-IV definition of AN included the idea of “refusal” to eat which implies intention. In DSM-5 this has been removed and the criterion now focuses more simply on individuals’ behaviours (see e.g., Becker, Eddy & Perloe, 2009). Another substantial change has been to exclude from the list of criteria that an individual needs to have amenorrhea. In part this was due to the difficulties of applying the criterion to young

(pre-pubertal) people and older (post-menopausal) women. In addition, there were numerous instances of individuals fulfilling all the criteria for AN apart from amenorrhea. Lastly, the time frames for bingeing and purging behaviours for the two main subtypes of AN are more clearly defined in DSM-5.

2. Bulimia Nervosa. The DSM-5 criteria include:

- A. Recurrent episodes of binge eating. An episode of binge eating is characterized by both of the following:
 - a. Eating, in a discrete period of time (for example, within any 2-hour period), an amount of food that is definitely larger than most people would eat during a similar period of time and under similar circumstances.
 - b. A sense of lack of control over eating during the episode (for example, a feeling that one cannot stop eating or control what or how much one is eating).
- B. Recurrent inappropriate compensatory behaviour in order to prevent weight gain, such as self-induced vomiting, misuse of laxatives, diuretics, or other medications, fasting or excessive exercise.
- C. The binge eating and inappropriate compensatory behaviours both occur, on average, at least once a week for 3 months.
- D. Self-evaluation is unduly influenced by body shape and weight.
- E. The disturbance does not occur exclusively during episodes of anorexia nervosa.

Bulimia nervosa has now lost the subtype criteria that used to exist in DSM-IV. It was felt that subgrouping bulimic patients served no useful clinical purpose. It was

also recognized that the non-purging subtype of bulimia was in practice indistinguishable from binge eating disorder (see e.g., Vervaeke, van Heeringen & Audenaert, 2004). Lastly, in DSM-5, the required frequency for bingeing episodes has been reduced compared to DSM-IV.

3. Binge Eating Disorder. The DSM-5 criteria include:

- A. Recurrent episodes of binge eating. An episode of binge eating is characterized by both of the following:
 - a. Eating, in a discrete period of time (for example, within any 2-hour period), an amount of food that is definitely larger than most people would eat in a similar period of time under similar circumstances.
 - b. A sense of lack of control over eating during the episode (for example, a feeling that one cannot stop eating or control what or how much one is eating).
- B. The binge-eating episodes are associated with three (or more) of the following:
 - a. Eating much more rapidly than normal.
 - b. Eating until feeling uncomfortably full.
 - c. Eating large amounts of food when not feeling physically hungry.
 - d. Eating alone because of feeling embarrassed by how much one is eating.
 - e. Feeling disgusted with oneself, depressed, or very guilty afterwards.
- C. Marked distress regarding binge eating is present.
- D. The binge eating occurs, on average, at least once a week for three months.
- E. The binge eating is not associated with the recurrent use of inappropriate compensatory behaviour (for example, purging) and does not occur

exclusively during the course of anorexia nervosa, bulimia nervosa, or Avoidant/Restrictive Food Intake Disorder.

4. Lastly, the diagnostic criteria for ‘other eating disorders’ which used to be called Eating Disorders Not Otherwise Specified (EDNOS) in DSM-IV, have now been changed to **Feeding and Eating Conditions Not Elsewhere Classified (FECNEC)** in DSM-5. The criteria are:

- A. **Atypical Anorexia Nervosa** - All of the criteria for anorexia nervosa are met, except that, despite significant weight loss, the individual’s weight is within or above the normal range.
- B. **Subthreshold Bulimia Nervosa (low frequency or limited duration)** - All of the criteria for bulimia nervosa are met, except that the binge eating and inappropriate compensatory behaviours occur, on average, less than once a week and/or for less than for 3 months.
- C. **Subthreshold Binge Eating Disorder (low frequency or limited duration)**
- All of the criteria for Binge Eating Disorder are met, except that the binge eating occurs, on average, less than once a week and/or for less than for 3 months.
- D. **Purging Disorder** - Recurrent purging behaviour to influence weight or shape, such as self-induced vomiting, misuse of laxatives, diuretics, or other medications, in the absence of binge eating. Self-evaluation is unduly influenced by body shape or weight or there is an intense fear of gaining weight or becoming fat.

- E. **Night Eating Syndrome** - Recurrent episodes of night eating, as manifested by eating after awakening from sleep or excessive food consumption after the evening meal. There is awareness and recall of the eating. The night eating is not better accounted for by external influences such as changes in the individual's sleep/wake cycle or by local social norms. The night eating is associated with significant distress and/or impairment in functioning. The disordered pattern of eating is not better accounted for by Binge Eating Disorder, another psychiatric disorder, substance abuse or dependence, a general medical disorder, or an effect of medication.
- F. **Other Feeding or Eating Condition Not Elsewhere Classified** - This is a residual category for clinically significant problems meeting the definition of a Feeding or Eating Disorder but not satisfying the criteria for any other Disorder or Condition.

In addition to DSM-5, the International Classification of Diseases, 10th edition (ICD-10; World Health Organisation, 2010) is used to classify eating disorders. These two classifications are widely used, but do have multiple limitations.

1.2. Difficulties with diagnostic criteria

Fairburn and Cooper (2011) reported that in clinical settings it is common to see individuals who first meet diagnostic criteria for anorexia nervosa (AN), then for bulimia nervosa (BN) and then Atypical Anorexia. According to the DSM-5, these individuals have had three different psychiatric conditions. This movement between diagnostic categories is particularly common during the recovery process, with individuals who have had AN frequently passing through phases of BN or atypical

eating disorders (Fairburn & Harrison, 2003), to the extent that this pattern of change over time might even be considered normal. The movement between the diagnostic categories can be in any direction, though some patterns are said to be more common than others.

Not only is there a problem with patients moving between diagnostic categories, but there is also a considerable degree of overlap between the categories themselves (e.g., Garfinkel, Kennedy & Kaplan, 1995). This leads to marked variability in the diagnoses applied to individuals and has contributed to the continued need to update the DSM criteria over time. Despite this uncertainty, many of the clinical features and behavioural patterns are shared across different eating disorders (e.g., Fairburn & Cooper, 2011; Fairburn & Harrison, 2003). The common aspects in all eating disorders seem to be related to over-evaluation of body shape and weight as well as extreme dieting. The main differences relate to the presence or not of binge eating and compensatory behaviours, or alternatively undereating and the consequences for weight status (Fairburn & Cooper, 2011). In addition to the shared psychopathology (Fairburn & Harrison, 2003), the mechanisms for development of eating disorders seem to be more similar than they are different (Fairburn, Cooper & Shafran, 2003). For example, twin studies suggest that a common set of genetic and environmental factors enhance the risks of developing any eating disorder (Bulik, Thornton, Root, Pisetsky, Lichtenstein et al., 2010; Strober, Freeman, Lampert, Diamond, & Kaye, 2000).

In the face of diagnostic uncertainty, several authors have tried statistical methods such as latent class analysis to attempt a symptom-based classification of eating disorders (see e.g. Keel, Brown, Holland & Bodell, 2012; Swanson, Horton, Crosby, Micali, Sonnevile et al., 2014; Wonderlich, Joiner, Keel, Williamson &

Crosby, 2007). However, these attempts have been confounded by the problem that the symptoms themselves that patients experience can change depending on the stage of recovery for an individual (Clausen, 2004).

Co-morbidity based approaches have also been suggested (see e.g., Wildes & Marcus, 2013). The idea is that a particular co-morbidity pattern can predict both the symptoms of an eating disorder and its maintenance (Stice & Agras, 1999; Westen & Harnden-Fischer, 2001). However, since co-morbidity based classifications do not necessarily include eating behaviours in their criteria, they could in principle include an even wider range of patients than was originally intended.

Yet another level of difficulty with the DSM-5 definitions for eating disorders is the poor agreement with results from clinical neuroscience (e.g., Park, Godier & Cowdrey, 2014; Steinglass & Walsh, 2006) as well as the fact that there is no simple mapping between the diagnostic criteria and treatment strategies and outcomes (e.g., Insel, Cuthbert, Garvey, Heinssen, Pine et al., 2010; Widiger & Samuel, 2005). This is particularly true for the atypical eating disorders, which are the diagnoses most commonly assigned, as compared to anorexia nervosa or bulimia nervosa (e.g., Fairburn & Harrison, 2003; Keel et al., 2012; Wonderlich et al., 2007) which are more clearly defined disorders, and where the treatment options are better validated.

Taking a pragmatic point of view, and with the aim of making clinical services more easily available to the greatest number of sufferers, Walsh and Sysko (2009) proposed the Broad Categories for the Diagnosis of Eating Disorders (BCD-ED). This proposal essentially relaxes some of the diagnostic criteria in the deliberate attempt to cast a wider net for the benefit of patients. In Fairburn and

Cooper's (2011) sample of 167 underweight participants, this strategy would lead to a 25% increase in individuals diagnosed with anorexia nervosa, and a 14% increase in individuals diagnosed with bulimia nervosa.

Fairburn and Cooper (2011) have pursued this line of thinking even further by proposing either that: a) all the additional eating disorders should be named "mixed eating disorders" but that anorexia nervosa and bulimia nervosa should be left as they are, or b) patients should be classified based entirely on clinical features with a focus on the presence or absence of bingeing, consistent with the transdiagnostic theory for treatment of eating disorders (Fairburn et al., 2003). Fairburn and Cooper (2011) rationalise this approach because: (i) the psychopathology across eating disorders is so similar (e.g., Fairburn & Harrison, 2003); (ii) at the early stages of an eating disorder, the attempts to control eating are largely successful, whereas when an individual gets older and has had the disorder for longer, they are more likely to lose control, and thus move diagnostic category even though there is no change in their psychological state (Fairburn & Cooper, 2011); and (iii) the core treatment targets are pretty much the same in all eating disorders, even though the treatments themselves may be different for different disorders (Fairburn et al., 2003; Fairburn, Cooper, Doll, O'Connor, Bohn et al., 2009).

While consolidation of the criteria for eating disorders across categories may prove helpful diagnostically, it may not help treatment because anorexia nervosa and bulimia nervosa are often managed differently. For example, drug treatment has proven beneficial for bulimia nervosa, but not so much for anorexia nervosa (Bulik, Berkman, Brownley, Sedway & Lohr 2007; Shapiro, Berkman, Brownley, Sedway, Lohr et al., 2007). Likewise, Cognitive Behavioural Therapy has been shown to

benefit individuals with bulimia nervosa much more than individuals with anorexia nervosa (Bulik et al., 2007; Shapiro et al., 2007). Thus, any new specification for diagnostic regimes may need to retain distinctions which have proven to be meaningful for patient management.

In summary, the diagnostic criteria currently used, while workable are arguably far from perfect. Transdiagnostic theory may provide the basis for a solution in DSM-6 in order to improve the clinical management for sufferers.

1.3. Aetiology of the eating disorders

Eating disorders are multi-faceted, multi-determined psychiatric disorders resulting from a complex interplay between biological, psychological, and socio-cultural factors (Fairburn & Harrison, 2003; Polivy & Herman, 2002). Garner (1993) has proposed that the development of eating disorders can be broken down into levels, each of which can include biological, psychological and socio-cultural factors. According to Garner's model, an eating disorder develops through precipitating factors in an already predisposed individual. Furthermore, perpetuating factors will maintain the eating disorder. The majority of the research into the aetiology of eating disorders has focused on the predisposing factors. Identifying who is vulnerable for developing an eating disorder opens up opportunities to intervene. Research in this regard has focussed on the role of biological factors, particularly genetics (see e.g., Connan, Campbell, Katzman, Lightman & Treasure, 2003) and the 5HT2-A gene (Collier, Arranz, Li, Mupita, Brown et al., 1997). Of the precipitating factors, the impact of dieting has been widely explored (e.g., Butryn & Wadden, 2005; Polivy & Herman, 1985; Treasure & Collier, 2000;), and is currently

included in most aetiological models for eating disorders. Extreme dieting can lead to body dissatisfaction (Stice & Shaw, 2002) in an already vulnerable individual, and can cause biological, psychological and behavioural effects including: changes in neural reward systems (e.g., Kaye, Fudge, & Paulus, 2009), appetite circuitry (e.g., Keim, Stern & Havel, 1998), cortisol secretion (e.g., Anderson, Shapiro, Lundgren, Spataro & Frye, 2002), changes in cognitive capabilities (e.g., Vreugdenburg, Bryan & Kemps, 2003), mood (e.g., Wells, Read, Laugharne & Ahluwalia, 1998) and obsessional tendencies (e.g., Keys, Vivanco, Minon, Keys & Mendoza 1954; Warren & Cooper, 1988). However, more recently, research efforts have focused on the role of body image dissatisfaction as the main precipitator for eating disorders, instead of dieting (Johnson & Wardle, 2005; Ricciardelli & McCabe, 2001; Stice, 2001). Currently, body image distortion together with starvation induced changes in physiological function like metabolic rate and psychological function like flexibility and cognition are seen as main maintenance factors for eating disorders (Carter, Blackmore, Sutandar-Pinnock & Woodside, 2004; Fairburn, Cooper, Doll & Welch, 1999; Pike, 1998; Strober, Freeman & Morrell, 1997).

1.3.1. Genetic influences

Family studies and twin studies have suggested that the risk of developing an eating disorder can be transmitted through the family line (e.g., Stober et al., 2000). For example, the incidence of AN in the first-degree relatives of AN probands has been found to be six times greater than that in the relatives of non-eating disordered controls (Gershon, Schreiber, Hamovit, Dibble, Kaye, et al., 1984). However, estimates of the heritability of AN based on twin studies are very variable, ranging from 33% to 84% (Bulik et al., 2010; Klump, Miller, Keel, McGue, & Iacono, 2001; Kortegeard, Hoerder, Joergensen, Gillberg & Kyvik, 2001). Interestingly, this

situation is similar to other complex medical, often neurological, conditions like Parkinson's disease and developmental dyslexia, where family studies have revealed strong epidemiological evidence for familiarity, but monozygotic/dizygotic twin comparisons produce much weaker evidence for heritability (Tanner, Ottman, Goldman, Ellenber, Chan, et al., 1999). Nevertheless, the fact that AN has been shown to be heritable has triggered a large number of genetic studies aimed at identifying genetic factors that increase the risk of the condition. Most of the recent investigations have targeted the control mechanisms for appetite and energy regulation, with a specific focus on the role of the hypothalamus. Before outlining some of these findings, we give a very brief overview of the physiology of appetite and weight control. This is necessarily over-simplistic; a comprehensive review is beyond the scope of this thesis.

The control of appetite, energy homeostasis and body weight is complex. It relies on dynamic interactions between the central nervous system (CNS) and a number of peripheral organs where energy is stored (e.g., in adipose tissue and the liver), to ensure sufficient and sustained energy levels for given circumstances. The CNS is continually updated about the state of: (i) food that is being / has been consumed, (ii) the energy stores and any state of change in critical organs, (iii) basal level and task dependent metabolic requirements. In return, the CNS controls: (i) tissues like the liver, adipose and the muscles that play key roles in energy homeostasis, (ii) the secretion of critically important hormones that modulate energy metabolism via the autonomic nervous system.

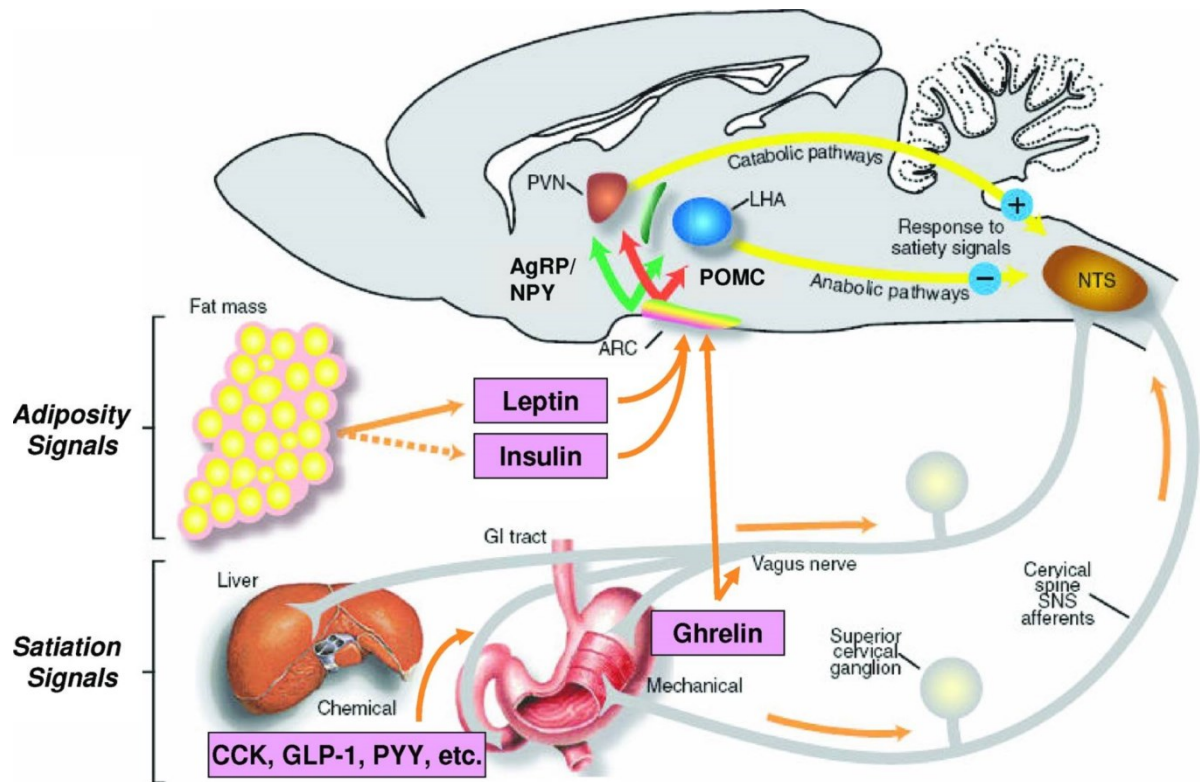


Figure 1.1: Overview of energy homeostasis in rat. PVN = paraventricular nucleus (hypothalamus); LHA = lateral hypothalamic area; ARC = arcuate nucleus (hypothalamus); NTS = nucleus tractus solitarii; AgRP = agouti-related protein; NPY = neuropeptide Y; POMC = pro-opiomelanocortin; CCK = Cholecystokinin; GLP-1 = Glucagon-like peptide; PYY = pancreatic peptide YY3-36. Adapted from Clarke, Weiss & Berrettini (2012).

Figure 1.1 outlines more specific details of the pathways involved. There are two major groups of signalling routes: those indicating the state of satiety and separately, those indicating adiposity. When we eat, the distension of the stomach, together with signals such as GLP-1 and CCK that are secreted from the stomach and intestine, induce neural responses in sensory afferents that travel to the hypothalamus, in particular. Together, these sources of information indicate the state

of satiety. Autonomic nervous system signals related to the state of satiety synapse with neurons in the NTS where they can have an influence on meal size. The stomach secretes Ghrelin and this also acts on vagal afferents as well as directly stimulating neurones in the ARC. With respect to adiposity, the hormonal signals related to body fat content, e.g., insulin and leptin, are carried in the blood stream to the brain, where they cross the blood-brain barrier. There they are detected by neurones in the ARC which then modulate the synthesis of the AgRP, POMC and NPY neuropeptides. Neurones in the ARC project to other nuclei in the hypothalamus, such as the LHA and PVN. Broadly speaking, the resulting drive from the PVN amplifies the consequences of satiety and leads to a catabolic state. By contrast, LHA output tends to suppress satiety, leading to an anabolic state. Therefore, by means of a complex, and carefully balanced feedback system, body fat tends to remain in an equilibrium state in the long term essentially by pitting energy requirement against meal size.

One way to investigate putative genetic influences on the appetite and weight control circuitry in AN is to carry out a candidate gene association study (CGAS). The rationale of this design is that a group of patients who all have the same condition should also have the same set of genetic mutations that increase the risk for the condition. Therefore, researchers measure the frequency of genetic polymorphisms in samples of patients and compare these to the frequencies observed in groups of control participants, who do not express the same phenotype. If a statistically significant difference in allele frequency is observed between the two groups, the usual inference is that the gene(s) involved may contribute to the aetiology of the disease in question.

One example of a relatively recent CGAS tested for the presence of polymorphisms in the appetite stimulating gene coding for AgRP (Vink, Hinney, van Elburg, van Goozen, Sandkuijl, et al., 2001). If AgRP is injected into the ARC, food intake is chronically increased for up to a month. Insulin and leptin have an overall effect to suppress AgRP / NPY neurones in the ARC. In the study by Vink et al., (2001), variants of the gene coding for AgRP were sequenced in 100 patients with AN. The polymorphisms they identified were subsequently genotyped in a further 45 AN individuals and 244 controls. Vink et al. (2001) showed a significant association of AN with the Ala67Thr variant, in which alanine is changed to threonine at position 67 in the amino acid chain. In another example, neurones in ventro-medial hypothalamus (VMH) express brain-derived neurotrophic factor (BDNF). In mice, VMH-specific gene repression of BDNF leads to hyperphagia and obesity, but normal energy expenditure, consistent with a role for VMH BDNF in the control of appetite (Unger, Calderon, Bradley, Sena-Esteves & Rios, 2007). Moreover, heterozygous mice lacking a copy of the BDNF gene (+/-) display hyperphagia and obesity (Gray, Yeo, Cox, Morton, Adlam, et al., 2006). Findings like these therefore led researchers to ask whether the BDNF might contribute to the aetiology of AN. Accordingly, Ribasés, Gratacòs, Fernández-Aranda, Bellodi, Boni et al. (2005) carried out a family-based study on eight European populations and found that AN was associated with the Val66Met polymorphism. In summary, these examples of CGASs have suggested a role for AgRP and BDNF in the pathology of AN. Unfortunately, the findings from CGASs with patients who have AN “... resemble those for most complex biomedical diseases - initial intriguing findings diminished by the absence of rigorous replication.” (Boraska, Franklin, Floyd, Thornton, Huckins, et al., 2014).

Another methodology of major importance for discovering genetic risk factors for medical diseases is the genome-wide association study (GWAS). As the name implies, millions of single-nucleotide polymorphisms (SNPs) can be genotyped across the entire genome, allowing identification of potential genetic targets, without requiring a priori hypotheses about which genes might be involved. GWASs have been successfully employed to reveal risk loci for diseases such as Crohn's disease, bipolar disorder, and diabetes (Wellcome Trust Case Control Consortium, 2007). In 2014, Boraska et al. published the largest ever GWAS on AN. These authors carried out a global meta-analysis of the samples obtained from 14 countries, comprising 5,551 AN cases and 21,080 controls across discovery and replication data sets. No findings reached genome-wide significance, although suggestive results were found for two variants: rs9839776 in SOX2OT and rs17030795 in PPP3CA. Two additional suggestive, though non-significant, signals were found that were specific to European patients with AN. The authors conclude that their data strongly suggest that true findings exist but their sample, despite being the largest yet tested, was underpowered for robust detection. Nevertheless, the authors remained optimistic that even larger genotyped AN case-control samples ought to reveal genetically determined differences in biological pathways that give rise to AN, and which may therefore point the way to specific interventions.

1.3.2. Psychological influences

Multiple psychological factors have been investigated as potential predisposing factors for eating disorders. Most of the studies have been correlational in nature, and thus more research from longitudinal or intervention studies is needed before definitive conclusions can be drawn. Amongst the most intensively explored psychological factors are obsessionality (e.g., Wonderlich, Lilenfeld, Riso, Engel &

Mitchell, 2005), perfectionism (e.g., Bardone-Cone, Wonderlich, Frost, Bulik, et al., 2007; Halmi, Sunday, Strober, Kaplan, Woodside, et al., 2000), neuroticism (e.g., Bulik, Sullivan, Tozzi, Furberg, Lichtenstein, et al., 2006; Eggert, Levendosky & Klump, 2007; Podar, Hannus & Allik, 1999; Wilksch & Wade, 2004), impulsivity (Claes, Bijttebier, Mitchell, de Zwaan & Mueller, 2011) and self-esteem (Button, Sonuga-Barke, Davies & Thompson, 1996). Thornton and Russell (1997) found that 37% of their sample of Anorexic patients met the diagnostic criteria for obsessive-compulsive disorder (OCD), and more importantly 86% of these individuals had been diagnosed with OCD before they were diagnosed with an eating disorder. However, other studies, including the classical Minnesota semi-starvation study from 1950 (Keys, Brožek, Henschel, Mickelsen & Taylor, 1950), have shown that obsessive symptoms can develop as a consequence of starvation, and that these symptoms can recede with weight restoration (Pollice, Kaye, Greeno & Wltzin, 1997). While equivalent findings have been reported with respect to neuroticism (Crisp & Stonehill, 1972; Stonehill & Crisp, 1977) as well as depression and anxiety (Crisp, Harding & McGuinness, 1974), it remains an open question whether these psychological factors are a cause or consequence of eating disorders.

1.3.3. Environmental influences

Experimental, observational and epidemiological research has suggested a substantial role for environmental influences in the aetiology of eating disorders. There was a dramatic increase in the incidence of eating disorders during the 20th century (Keel & Klump, 2003) consistent with rapidly changing environmental factors. Cross-culturally, eating disordered symptomatology is more prevalent in North America and Western Europe than in Asian or Eastern European countries (Boyadjieva & Steinhausen, 1996; Lee, Rhee, Park, Sohn, Chung, et al., 1998), and

it has been shown that immigration to Western societies heightens the risk for developing eating disorders (e.g., Nasser, 1986). Urban areas have been found to have a higher prevalence of eating disorders than rural areas (e.g., Hoek, Bartelds, Bosveld & van der Graaf, 1995) and certain subgroups, like dancers, athletes and models who presumably share niche environments, have been shown to be more vulnerable to eating disorders (e.g., Garner & Garfinkel, 1980; Hamilton, Brooks-Gunn & Warren, 1985).

Specific environmental influences that have been investigated include: socio-cultural factors, particularly the role of the media (e.g., Harrison & Cantor, 1997; Stice, Schupak-Neuberg, Shaw & Stein, 1994), family focus on appearance (e.g., Davis, Shuster, Blackmore & Fox, 2004; Keel, Heatherton, Harnden & Hornig, 1997), and peer group influence (e.g., Wardle & Watters, 2004).

Frequency of exposure to images of thin bodies in the media is associated with the emergence of disordered eating behaviours and body dissatisfaction (Grabe, Ward & Hyde, 2008; Groesz, Levine & Murnen, 2002; Knauss, Paxton & Alsaker, 2007; Tiggemann, 2003; Tiggemann & McGill, 2004;). However, the direction of this relationship is not clear. One possibility is that exposure to advertising, magazines and social media may act as a precipitating cause for eating disorders, at least in some individuals (Levine & Murnen, 2009; Martinez-Conzales, Gual, Labortiga, Alonso, de Irala-Estévez, et al., 2003). For example, laboratory based studies have shown convincingly that exposure to an adapting stimulus, which is drawn from a continuous range of morphs between two faces of different identity, can dramatically shift facial recognition patterns in the direction of the adapting stimulus (Leopold, O'Toole, Vetter, & Blanz, 2001). Similar findings have been shown for the perception of body size. Winkler and Rhodes (2005) found that

participants' concept of what is normal and attractive was plastic. In an adaptation paradigm, they found that the most attractive and most normal looking bodies appeared narrower to observers after they had been exposed to narrow bodies. However, while the most normal looking bodies changed significantly after exposure to wide bodies, the most attractive body did not. Clearly, despite the evidence for some asymmetry in the adaptation effects, these results showed that, at least in the short term, perceptions of body shape can be influenced by experience and, by implication, the immediate environment. Moreover, what is considered to be an 'attractive' body shape has been shown in a number of cross-cultural studies to depend on the particular cultural environment (e.g., Tovée, Swami, Furnham & Mangalparsad, 2006). And there is also evidence that within a culture, these standards can shift. Historically, the optimal body size for 'beauty' may be changing. In the 1950s most Playboy models had a BMI around 19.5. By 2000, this had shrunk to 18 (Voracek & Fisher, 2002). Furthermore, the immediate context in which an individual is placed, for example whether they are surrounded either by larger or smaller individuals, has also been shown to influence that person's perceived body size (see e.g., Bateson, Tovée, George, Gouws & Cornelissen, 2014). Thus, it is not hard to imagine that the cultural milieu, together with the media's power to influence our visual diet, might have a strong adapting influence on the body image especially of adolescents, and that might in turn contribute to the development of eating disorders. Clearly prospective, longitudinal studies are required to test for an increase in the incidence of eating disorders in cultures that are only just starting to be exposed to westernized culture.

How might the environment within an individual's family affect the development of eating disorders? Family studies have investigated parental voiced

opinions about dieting and body image, parental dieting itself, family eating habits and family dynamics, particularly attachment style (e.g., Ward, Ramsay & Treasure, 2000). One consistent finding is that parental eating habits and parents' comments about their daughters' body size and shape have both been shown to predict daughters' body image satisfaction and disordered eating (e.g., Haworth-Hoepfner, 2000; Keel et al., 1997). One note of caution, however, is that many studies of this kind have not drawn a clear distinction between family influences, which are environmental, from heritable effects which are genetic. Outside of the family circle, a number of studies have also revealed peer group influences on the body satisfaction of adolescent girls in particular (e.g., Dohnt & Tiggemann, 2006; McCabe & Ricciardelli, 2001). Social comparison theory (Festinger, 1954) has been suggested as one mechanism by which peer influence might be internalised in these cases (Stormer & Thompson, 1996; Thompson & Heinberg, 1999). In general, there is a consensus that socio-cultural influences are likely to play a more substantial role in the development of BN than for AN (e.g., Stice et al., 1994).

1.3.4. Body Image Distortion (BID), an aetiological perspective

An important question is whether distorted body image, which is a cardinal feature of eating disorders, is a cause or consequence of the illness. In aetiological models of eating disorders, body dissatisfaction, which is a key component of body image distortion, is frequently placed on the precipitating axis (e.g., Polivy & Herman, 2002; Stice & Shaw, 2002) as a causal as well as a maintenance factor (e.g., Fairburn, 2008; Fairburn & Cooper, 1989). For example, it has been suggested that body dissatisfaction, driven by socio-cultural pressures (e.g., Stice & Shaw, 2002), may in turn lead individuals to severe dieting in order to control their body size and shape concerns (Stice & Shaw, 2002). It has also been suggested that body

dissatisfaction contributes to negative affect, which can lead to disordered eating in order to compensate for and provide a psychological diversion from emotional challenges (e.g., Stice, 2001). In support of a role for body dissatisfaction as a predisposing factor for eating disorders, studies have shown that elevated body size and shape concern in early childhood can predict eating disorders later in life (Agras, Bryson, Hammer & Kraemer, 2007). Perhaps not surprisingly, there are also studies which suggest that body dissatisfaction itself may be modulated by the course of an eating disorder (e.g., Stice & Shaw, 2002).

Cohen-Tovée (1993) investigated how mood induction techniques can influence body satisfaction. She found that lowering mood reduced body satisfaction scores particularly in groups of individuals who were already dissatisfied with their bodies. This interaction suggests that the consequence of a negative psychological state is likely to be more damaging to those who already have problems with body image. However, in a sample of 509 female participants, Heatherton, Mahamedi, Striepe, Field and Keel (1997) showed that when youngsters moved into adulthood their disordered eating behaviours reduced, but their body dissatisfaction increased, suggesting a reverse relationship between the two. Similar results were found by Keel et al. (2007). Therefore, it is quite possible that body image distortion in eating disorders may be both a cause and a consequence. At the very least, the data suggest a complex picture of interacting influences over time.

An important question is when, in time, does BID develop. Garner and Garfinkel (1982) proposed that early developmental deviation may distort body image, leading ultimately to eating disorders. Crisp (1997) and Crisp, Hall and Holland (1985) went as far as suggesting that any maturational delays combined with genetic, socio-cultural and psychological predisposing factors might lead to the same

outcome. A child's body image develops hand in hand with their cognitive abilities. Different biological, cultural and individual psychological factors influence different stages on this developmental trajectory. Infants first achieve self-perception and recognition (i.e. "this is me"), which, with time, develops further into self-representation (i.e. "what are the features that make me") as a toddler. Self-other comparisons (i.e. "what differentiates me from others") start to emerge at school age and self-image (i.e. "how do I compare with others and how do I feel about it") together with ideal body image develops approximately at the age of 8 (Smolak & Levine, 2001). Individual physical development (see e.g., Stice & Shaw, 2002; Smolak, 2004), gender norms (e.g., Smolak, 2004), and social learning (e.g., Murnen, Smolak, Mills & Good, 2003) also have their effects on this developmental curve. A number of authors have therefore suggested that the roots of distorted body image may lie in adverse early childhood experiences (e.g., Jackson, Grilo & Masheh, 2000; Smolak & Levine, 2001), such as teasing (Thompson, Coover, Richards, Johnson & Cattarin, 1995).

An alternative proposal is that body image is most likely to become distorted when individuals move from childhood to adulthood; it may be no coincidence that most cases of eating disorders start in adolescence (Hoek & Van Hoeken, 2003). A number of studies have investigated the relationship between chronological age and body image preferences (Cohn, Adler, Irwin, Millstein, Kegeles et al., 1987; Gardner, Friedman & Jackson, 1999), and it has been shown that at around the age of 13, girls want to have much thinner bodies (e.g., Gardner et al., 1999). It is also at this age that children are increasingly exposed to the kinds of social influences which can trigger negative body image (Thompson & Zuroff, 1999). At this age youngsters are arguably more suggestible, may experience expectations for gender norms,

sexual harassment and objectification and are often lacking models which permit acceptance of diversity in body weight and shape (e.g., Levine & Smolak, 2001). These factors combined with pubertal weight and body shape change, loosened parental ties and increased autonomy can modulate body satisfaction. Social reinforcement, social comparison and modelling are likely to be working through peers' influence, rather than family at this stage. There is particularly strong evidence that social comparison can trigger body dissatisfaction and motivate eating disordered behaviours in adolescence (Dittmar & Howard, 2004). Furthermore, as body schemata are still developing in adolescence, at this vulnerable stage depression or dieting triggered by body dissatisfaction can pave the way to disordered eating (Stice, 2001).

In summary, the aetiology of eating disorders is multi-factorial and complex, and the contribution of various factors is at best incompletely understood. A particular challenge is to understand the way genetic makeup and environmental factors interact.

1.4. Neural correlates of eating disorders

Neuroimaging methodology has found its way into almost every domain of cognitive, experimental and clinical psychology. Inevitably this has proved to be the case with the eating disorders. Therefore we give a brief outline of some of the neuroimaging literature in this field, with a particular focus on body image perception and BID.

To date neuroimaging studies in the arena of eating disorders have focused largely on: mapping changes in cortical volume of people with AN compared with non-eating disordered controls (e.g., Katzman, Lambe, Mikulis, Ridgely, Goldbloom et al., 1996; Mohr, Zimmermann, Roder, Lenz, Overbeck et al., 2010); identifying the neurobiological correlates of anorexic behaviour (e.g., Kaye, Bailer, Frank, Wagner & Henry, 2005; see also Kaye, Fudge & Paulus, 2009); mapping pathophysiological changes in the reward networks (e.g., Kaye et al., 2009; Keating, 2010; Schultz, 2004) and the cortical circuitry for appetite control in patients with AN (e.g., Santel, Baving, Krauel, Münte & Rotte, 2006; Uher, Murphy, Friederich, Dalgleish, Brammer et al., 2003). Whilst multiple studies have identified both structural and functional changes in occipito-temporal cortices related to visual processing in AN participants (e.g., Suchan, Busch, Schulte, Gronemeyer, Herpertz et al., 2010; Uher et al., 2003), it is not clear whether the changes are specific to body size perception, or a result of starvation. Favaro, Santonastaso, Manara, Bosello, Bommarito et al. (2012) also proposed that distorted body size perception may be caused by failure to integrate visual and somatosensory information adequately.

In a systematic review, Gaudio and Quattrocchi (2012) point out that while the behavioural literature has emphasized the multidimensionality of AN, most neuroimaging studies fail to take this into account, and have focussed on – usually – just one dimension in each study. Therefore, Gaudio and Quattrocchi (2012) propose that this overly simplistic approach may be responsible for the poor consistency of the findings between studies in this field. In an attempt to reveal some order, they reviewed 12/88 fMRI studies, which were produced up until the end of 2011. To be eligible for inclusion in the review, the studies had to have investigated a sample of

patients with AN in cross-sectional, case-control or longitudinal design or to have investigated a sample of healthy subjects, and to have used tasks measuring the whole body image. Gaudio and Quattrocchi (2012) sought evidence for neural correlates of three aspects of body image: perceptive (defined by viewing photos of the participants' own and/or other's bodies, or line drawings), affective (defined by viewing distorted versions of participants' own bodies, or slim other people's bodies, or viewing/listening to unpleasant words regarding participants' own bodies), and cognitive (where experimental tasks elicited beliefs about body shape and appearance, but were not based on viewing images of bodies). The key regions which discriminated between participants with AN and healthy controls on these three dimensions are summarized in Figure 1.2 below.

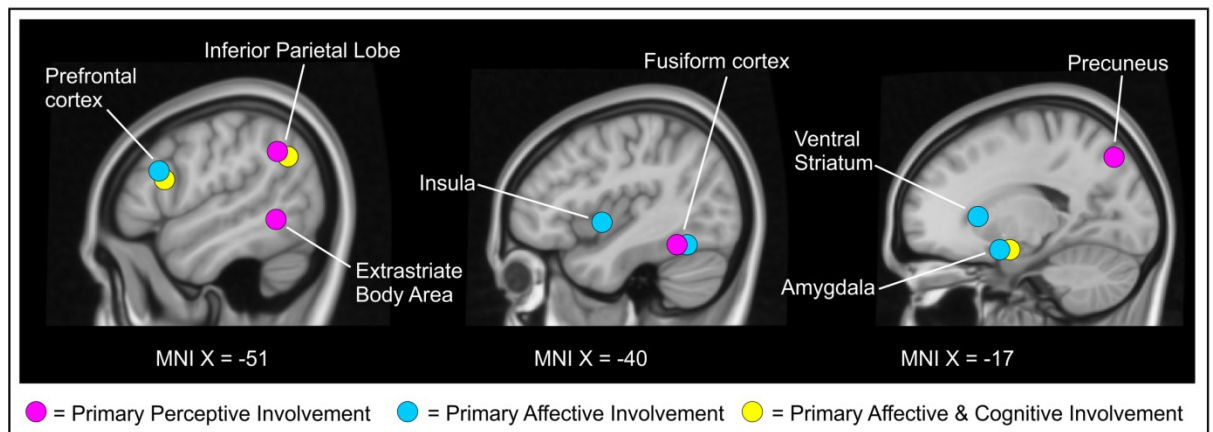


Figure 1.2: Left hemisphere MR sagittal slices, at three MNI x-coordinates, showing the main sites for differences between patients with AN and healthy controls from structural and functional MR studies, as summarised in Gaudio and Quattrocchi (2012).

According to the categorization drawn up by Gaudio and Quattrocchi (2012), the neuroimaging data for the perceptive component of BID was probably the most

consistent across studies. Specifically, structural and functional data from the parietal cortex (precuneus and inferior parietal lobe) suggested that cortical processing in these regions was related to impaired body size estimation in AN (e.g., Mohr et al., 2010). However, results from the extrastriate body area with respect to self-body image identification in AN patients (Sachdev, Mondraty, Wen & Gulliford, 2008; Vocks, Busch, Gronemeyer, Schulte, Herpertz et al., 2010a, 2011) as well as healthy subjects (Hodzic, Muckli, Singer & Stirn, 2009; Vocks, Busch, Gronemeyer, Schulte, Herpertz et al., 2010b) were equivocal.

With respect to the affective component of body image, unpleasant stimuli (i.e. images of self and others made to look fat) evoked smaller blood oxygen level dependent (BOLD) responses in the prefrontal cortex (PFC) of AN patients compared to controls, and this was associated in some studies with hyperactivation of the amygdala. Stimuli that were pleasant/desirable (i.e. images distorted to look thin) or aversive (i.e. comparing self to slim fashion model images) elicited differences between AN patients and controls in the insula and the PFC. Moreover, Fladung, Grön, Grammer, Herrnberge, Schilly et al., (2010) also showed interactions between the responses to pleasant stimuli and participants' emotional responses in the ventral striatum. In short, stimulus conditions defined as aversive modulated neural responses in different cortical areas compared to perceptive stimuli, suggesting (like most other cognitive tasks) the involvement of a distributed network for processing body image judgements, with different cognitive subcomponents potentially 'assigned' to different subcomponents of the network.

Gaudio and Quattrocchi (2012) concede that the evidence to support a separate cognitive component for body image distortion was weak. They suggest that dysfunction of the parietal cortex might disturb beliefs about one's own body image

and its relation to the perceptive component, but conclude that further studies are required. For example, like Williamson, Muller, Reas and Thaw (1999), they suggest comparing the cortical responses to pleasant, unpleasant and neutral words about body image to neutral words that are unrelated to body image.

Finally, a recent approach to investigating involvement of the insula in AN has been explored by Bär, de la Cruz, Berger, Schultz and Wagner (2015), by measuring the cortical responses to pain. It has been proposed that many of the symptoms of AN might be caused by a failure to integrate and regulate autonomic, sensory and affective stimuli (Nunn, Frampton, Gordon & Lask, 2008). Since the insula has a distributed pattern of connections between the frontal, temporal and parietal cortices (Augustine, 1996), it is able to play a pivotal, integrative role in the processing of bodily homeostasis, emotion, interoception, and pain (Craig, 2002, 2009). Therefore, demonstrating impaired structural and/or functional integrity of the insula, indexed by abnormal pain processing, could go some way towards accounting for the symptomatology of AN.

1.5. Body Image Distortion (BID), definition, mechanism and measurement

Body image distortion, one of the central criteria for all eating disorders, has been shown to predict the severity and persistence of the condition (Fairburn & Harrison, 2003), the likelihood of relapse (Channon & De Silva, 1985) and can be found even in those individuals who have otherwise made a full recovery (Lautenbacher, Kraeche & Krieg, 1997). Furthermore, body image distortion is very hard to treat (Treasure, Claudino & Zucker, 2010) and there are very few treatment methods which are specifically targeted at body image distortion.

Slade (1988) defined body image as a picture we have in our minds about our body size and shape and the feelings related to that image. The current consensus is that body image is a multidimensional construct consisting in cognitive-behavioural, perceptual, emotional, evolutionary, neuroscientific, and sociocultural factors (Cash & Smolak, 2011). The possibility that an individual's body image may be disturbed arises when that 'image' diverges from reality and the feelings related to this mismatch have become overwhelming for that individual. Body image distortion (BID) can be seen in eating disorders and other weight disorders, like obesity, which is currently not labelled as a disorder in DSM 5. It is important to distinguish BID from Body Dysmorphic Disorder (BDD), which is a separate and distinct condition in which an individual becomes obsessed with a(some) flaw(s) in their body. There may or may not be a real physical feature around which the obsession develops. Whereas the focus of concern in BID tends to be the whole body, individuals with BDD tend to focus their concern on, and obsess about one part of the body.

1.5.1. Components of BID

In an influential meta-analysis, Cash and Deagle (1997) consolidate the idea that body image distortion has two independent modalities: perceptual body-size distortion and attitudinal or cognitive-evaluative body dissatisfaction. (Note: more recent reviews exist, e.g., Skrzypek, Wehmeier & Remschmidt (2001), but come to essentially the same conclusion). Perceptual distortion refers to an individual having difficulty accurately gauging their body size. Attitudinal dissatisfaction refers to feelings of unease and distress about body size, shape or appearance. An alternative division in to cognitive (i.e. what someone thinks they look like) versus affective (i.e. how someone feels about what they look like) distortion was proposed following early work by Crisp and Kalucy (1974). However, this distinction is

thought to be equivalent to the division drawn by Cash and Deagle (1997). A key finding from Cash and Deagle's (1997) meta-analysis is that the comparisons between eating disordered patients and non-eating disordered controls showed substantially larger differences (index by Hedges and Olkin's *d* statistic) for the tasks that they labelled as attitudinal compared to perceptual (by a factor of ~2).

1.5.2 Measuring the attitudinal aspect of BID

As pointed out by Cash and Deagle (1997), the attitudinal component of body image is often referred to in terms of body dissatisfaction or disparagement. As discussed earlier, most multifactorial models of the eating disorders assign body dissatisfaction a major causal role (e.g., Stice, 2001). Indeed, some computational models of eating disorders attempt a very fine grained partitioning of the various influences that behavioural and social influences, including body dissatisfaction, contribute to their aetiology. See, for example, a version of the Tripartite Influence model that attempts to explain the emergence of bulimic behaviour in Figure 1.3 overleaf (Thompson, Heinberg, Altabe & Tantleff-Dunn, 1999; van den Berg, Thompson, Obrowski-Brandon & Covert, 2002). This model was derived from covariance structure modelling of ~30 separate measures from 196 female undergraduate participants. However, for the purposes of this thesis, our strategy is to focus primarily on the perceptual component of body image distortion and to pit this against attitudinal factors. Therefore, we need to justify an approach which will suffice in terms of accounting for (most of) the variance related to body dissatisfaction, without having to maintain the level of complexity illustrated in Figure 1.3 overleaf.

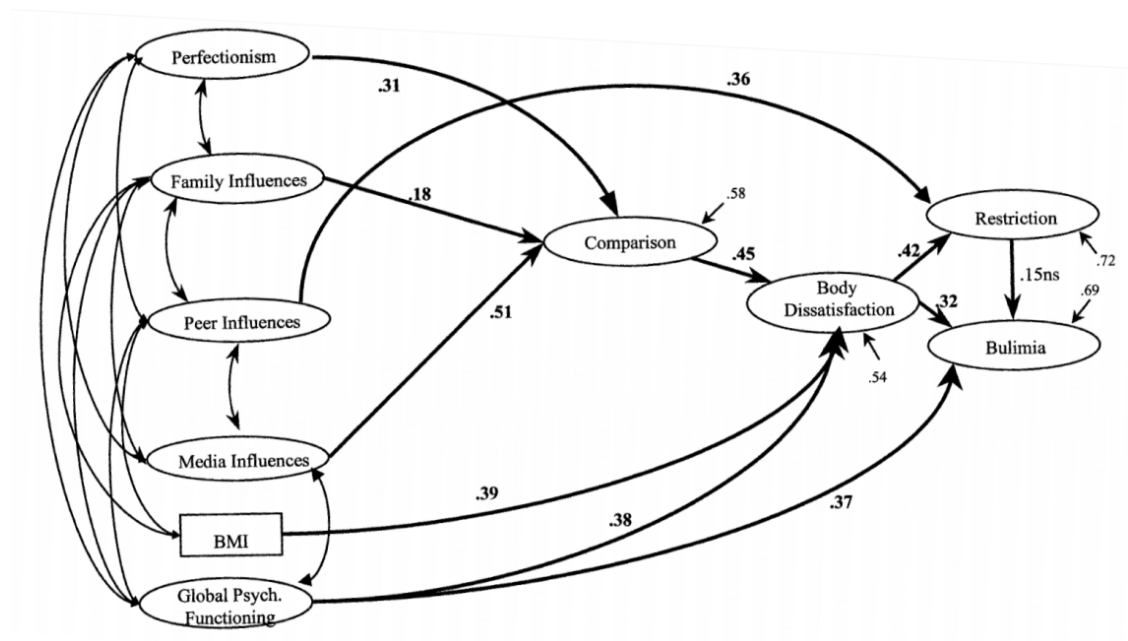


Figure 1.3: Tripartite model of the emergence of bulimia nervosa from van den Berg et al. (2002).

1.5.2.1. Body dissatisfaction

Body dissatisfaction has been conceptualized as a mis-match between an individual's actual versus their ideal weight/shape. In order to assess this, participants may be asked to select or adjust an image to illustrate their preferred body size or shape. This approach can therefore quantify the extent of any discrepancy between a person's perception of self versus their ideal (see e.g., Cooper & Taylor, 1988; Szymanski & Cash, 1995), and it is a measurement we use in Experiment 4, Chapter 5.

However, it is probably more common to measure body dissatisfaction by asking people specific questions about how satisfied/dissatisfied they are with their bodies or body parts, by means of a self-report questionnaire or structured interview (see Ben-Tovim & Walker, 1991; Cash & Brown, 1987). Secord and Jouard (1953)

developed one early example of this approach with their Body Cathexis Scale in which participants indicated how satisfied they were with forty 40 body sites and functions. Questionnaires that have been designed more recently however, tend to focus on whole body satisfaction, in a holistic way, instead of estimating a variety of discrete attributes from which a (weighted) combined measure of body dissatisfaction can be calculated (see e.g., Brown, Cash & Mikulka, 1990).

The Eating Disorder Examination Questionnaire (EDE-Q; Fairburn & Beglin, 1994) is one such example. It is a widely used psychometric tool used to assess and diagnose eating disorders. It was derived from The Eating Disorder Examination interview (EDE; Fairburn & Cooper, 1993) which is, arguably, one of the instruments of choice for clinicians. The EDE-Q is a self-report questionnaire, comprising 36 items, which provide a wide ranging assessment of eating-disordered behaviour. Two of the four psychopathological domains that the EDE-Q assesses fall squarely within the domain of body dissatisfaction: i.e. body shape and weight concerns. A number of studies have demonstrated a high degree of agreement between EDE-Q and EDE assessments of the same individuals, thus indicating that moving away from a formal interview structure, conducted by a trained interrogator, to a self-report format is valid both for members of the general population (e.g., Mond, Hay, Rodgers, Owen, & Beumont, 2004) as well as patient samples for the conditions bulimia nervosa and binge eating disorder (Carter, Aime, & Mills, 2001; Grilo, Masheb, & Wilson, 2001). The EDE-Q has also been shown to have good internal consistency and test-retest reliability (Luce & Crowther, 1999; Mond, Rodgers, Hay, Owen, & Beumont, 2004).

The Body Shape Questionnaire, first reported Cooper, Taylor, Cooper & Fairburn (1986), is another self-report assessment of weight and shape concerns. It

has also been shown to have good test-retest reliability as well as ‘concurrent validity’ when compared to other measures of body image (Rosen, Jones, Ramirez & Waxman, 1996). It is particularly convenient to administer as part of a test battery, since validated short forms have been produced (Evans & Dolan, 1993).

1.5.2.2. Self-esteem, mood and depression

Self-esteem with respect to body weight has been shown to be reduced in patients with eating disorders (Geller, Johnston, Madsen, Goldner, Remick et al. 1998). Moreover, longitudinal studies have found evidence that adolescent females who have low self-esteem are at increased risk of developing eating disorders within a few years (Button et al., 1996). Furthermore, Heatherton & Polivy (1992) found that dieting may also lead to reductions in self-esteem that contributed specifically to disordered eating.

Mood disorders, notably depression, and eating disorders frequently occur in the same individual. In principle, this co-morbidity may arise as a result of a primary disturbance of mood in eating disorders, or it may occur as a secondary consequence. A third possibility is that both conditions may be caused in the same individuals by the same common psychosocial or biological (e.g., genetic) factors. However, Stice, Hayward, Cameron, Killen and Taylor (2000) did find that eating disordered symptoms together with dietary restraint could predict the emergence of depression at a later point in time, in individuals who were not depressed when first tested. Therefore, in those individuals where depression and eating disorders co-occur, this is consistent with the idea that the eating disorders may need to have emerged first. Moreover, Steiger, Leung, Puentes-Neuman and Gottheil (1992) observed that not having a healthy body image may well be unique to the eating disorders.

Other studies suggest that depression and anxiety may be more state-dependent features that resolve when the symptoms of eating disorder resolve (Lehoux, Steiger, & Jabalpurlawa, 2000). Perhaps not surprisingly, other studies still have found evidence for a completely opposite temporal relationship: i.e. that eating disorders may follow on once mood disorder has already become established (e.g., Godart, Flament, Lecrubier & Jeammet, 2000). Nevertheless, since experiments which induce negative affect have been shown to increase body dissatisfaction and body size over-estimation in patients with bulimia nervosa (Kulbartz-Klatt, Florin & Pook, 1999), whether disordered mood follows disordered eating, or the other way around, may, in the end matter less than the fact that negative affect can contribute to eating disorder symptoms.

In summary, in order to estimate as much variance in the attitudinal component of distorted body image as possible for this thesis, we have chosen to administer self-report tasks that measure body dissatisfaction, tendency towards depression and self-esteem. We note that in a previous study, Cornelissen et al. (2013) demonstrated high levels of inter-correlations among such variables, to the extent that they were justified in using factor analysis to extract one, sometimes two, psychometric latent variables in order to index the attitudinal aspects of body image distortion.

1.5.3. Measuring the perceptual aspect of BID

A wide variety of methods has been used to estimate the perceptual dimension of body image distortion, starting from image marking procedures (Askevold, 1975) and moveable calliper techniques (Slade & Russell, 1973), through body-distorting mirrors (Traub & Orbach, 1964) and silhouette methods (Bell, Kirkpatrick & Rinn, 1986) to distorting photograph and video techniques (Gardner

& Moncrieff, 1988; Garner, Garfinkel, Stancer & Moldofsky, 1976; Probst, Vandereycken & Van Coppenolle, 1995; Shafran & Fairburn, 2002). In many early studies figure scales were used, where participants were asked to identify which figure they believed to be closest to their own. However, their use has largely fallen out of favour owing to the lack of validated psychometric properties (Gardner and Brown, 2010). More recent methods include applying body part morphs to photographs of individuals (e.g., Slade & Russell, 1973; Tovée, Benson, Emery, Mason & Cohen-Tovée, 2003) and the whole body video distorting technique (VDT; e.g., Allebeck, Hallberg & Espmark, 1976; Freeman, Thomas, Solyom & Miles, 1984; Gardner & Bokenkamp, 1996; Smeets, Ingleby, Hoek & Panhuysen, 1999). The distinction between whole body and body part methods, which has persisted throughout the literature, gave rise to concern about how comparable the results were from the two methodologies. Slade (1988) was first to point out that outcomes, reliability and sensitivity were often different, such that body part methods often gave rise to overestimates and whole body methods frequently led to underestimation. However, a formal meta-analytic comparison between these two approaches to body size estimation by Smeets, Smit, Panhuysen and Ingleby (1997) showed convincingly that both methodologies showed overestimation by participants with anorexia. While the average effect size was smaller for the whole body method, it showed far greater consistency than the body part method across studies.

In this thesis, we use a whole body method to measure participants' body size estimates. The principal behind most of the whole body measurement methods used to date has been to present participants with images of a body that have been expanded or contracted in the horizontal dimension, in order to simulate changes in adiposity. This has been achieved in a variety of ways. In the distorting photograph

technique, a slide of the person's body is projected on to a screen through a variable anamorphic lens. Participants adjust the width of the optically distorted image on the screen until it matches the size they believe themselves to be (Garner et al., 1976), and the percentage distortion of the image is recorded. This may be negative (under-estimation), zero (accurate perception) or positive (over-estimation). Equivalent methods using video (i.e. the video distorting technique, VDT) were developed by Allebeck, Hallberg & Espmark (1976), and these have even been extended to a life size screen distortion method (Probst, Vandereycken & Van Coppenolle, 1991).

As Cornelissen, Tovée & Bateson (2009b) have shown, a major problem with whole body image manipulations such as the VDT, which compress/expand the horizontal axis of the stimulus image, is that they amount to a 'multiplicative' model for representing body weight change – i.e. the horizontal width of the stimulus image is scaled multiplicatively. While this succeeds in changing the width of the body consistent with changing adiposity (Smith, Tovée, Hancock, Cox & Cornelissen, 2007), it fails to capture other structural changes in the abdomen, chest and limbs, and it comes at the expensive of introducing systematic distortions of body shape which do not occur in reality (Cornelissen et al., 2015). For example, as illustrated in Figure 1.4 overleaf, the width of the shoulders and the hips in particular change inappropriately. Moreover, features such as the width of the gap between the thighs, which normally would be expected to reduce with increasing adiposity, actually increases with increasing image expansion in the VDT. Finally, as shown by Cornelissen et al. (2009b), waist hip ratio (WHR) in UK females should show a monotonically increasing, decelerating relationship with BMI, yet the multiplicative model of fat deposition completely fails to capture this relationship.

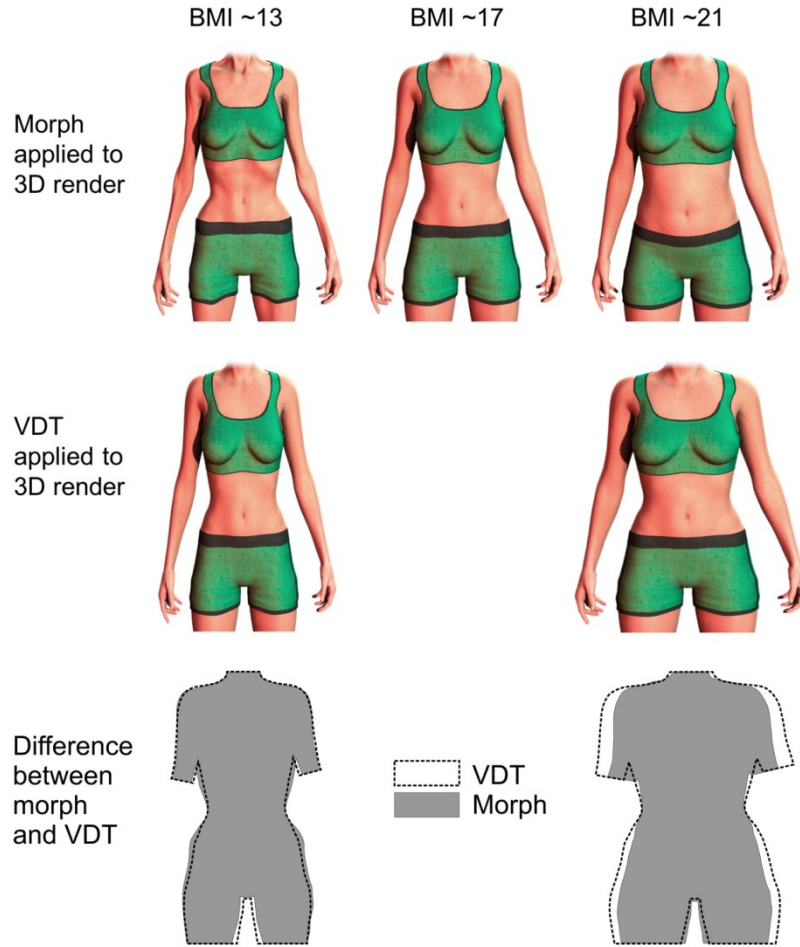


Figure 1.4. The top row shows three of our CGI stimuli representing BMI 13, 17 and 21 respectively. The images in the second row have had the video distortion technique (VDT) applied to the central image in the first row. To facilitate comparison between techniques, the waist widths of the compressed (left) and expanded (right) images on the second row correspond to those from the BMI 13 and 21 images from the top row, respectively. The third row shows a direct comparison between the outlines of the images produced by these manipulations. The CGI image is the solid grey outline and the VDT image is the dotted line.

For all these reasons, in this thesis we have developed a new whole body method to measure the perceptual aspect of body image distortion. The details of this method are given in the Methods Chapter 2. To do this we have used the same

computer-generated imagery (CGI) methods as the film and games industries to create 3D images representing a full spread of BMI. The principal advantages of this method are: (a) the identity of the person in the image is clearly maintained over a wide BMI range; (b) the images can be calibrated for BMI, and therefore a participant's body size estimate in BMI units can be compared with their actual body size in BMI units; (c) the 3D rendered stimulus images are high definition and photorealistic.

1.6. Measuring actual body size

It is probably a fair assumption that when researchers refer to perceptual distortion occurring when a person has difficulty accurately gauging her body size, what they mean is difficulty assessing adiposity. So, how should the adiposity or body fat of the participant be measured such that it can be compared to their own body size estimate?

Body fat can be measured in a variety of different ways: caliper measurements of skin fold thickness, bio-impedence, hydrostatic weighing, dual energy X-ray absorptiometry and the Body Mass Index (BMI). The “gold standard” for measuring body composition is dual energy X-ray absorptiometry (DXA) (Kohrt, 1998). This technique uses the relative absorbance of X-rays by the body to calculate fat content. Owing to its cost, and the non-availability to most researchers, this is not a viable choice. Arguably, the next best option is hydrodensitometry in which a participant's underwater weight and volume is measured and mathematically transformed to percentage body fat. However, this method can be confounded by subjects whose relative muscle mass is unusually high or low (Nelson, Fiatarone,

Layne, Trice, Economos et al., 1996), rendering anomalously high or low body fat respectively.

Amongst cheap and easily accessible methods should be included bioelectrical impedance analysis (BIA). Participants stand on a set of scales and the impedance (i.e. $Z^2 = R^2 + X_c^2$, where Z = impedance, R = resistance and X_c = reactance) experienced by a weak alternating electrical current passing from one foot, through the body, to the other, is measured. Impedance is higher when the ratio of body fat to body water is higher, hence can be used to estimate body fat. However, varying hydration levels in the body can alter substantially the impedance, thereby changing the apparent amount of body fat (Thompson, Thompson, Prestridge, Bailey, Bean et al., 1991). Another problem with this method is the differences in fat deposition between males and females (Power & Schulkin, 2008). Males carry most of their fat in their abdomen in the form of visceral fat which BIA cannot 'see' (Lohman, Caballero, Himes, Davis, Stewart et al., 2000). By comparison, females carry most of their body fat on their thighs as subcutaneous fat (Lemieux, Prudhomme, Bouchard, Tremblay & Despres, 1993). These sex differences therefore need also to be taken into account. In addition, BIA has been shown to underestimate total body fat mass when compared to DXA, especially in those with a total body fat above 25% (Leahy, O'Neill, Sohun, & Jakeman, 2012).

Another cheap, though more time consuming method of body fat measurement is the skinfold thickness method (SFT). This involves measuring pinches of skin using Harpenden callipers at 3 to 9 anatomical points on the body. These pinches are usually taken from just one side of the body, with the callipers being used 2-3 times on each point of the body in order to achieve an average measurement (for reliability). Durnin and Womersley (1974) suggested that 4 points

on the body (triceps, biceps, subscapular and suprailiac crest) can provide accurate estimates. However, SFT only measures subcutaneous fat and is therefore a poor predictor of abdominal fat mass and is likely to underestimate the body composition in obese individuals (Watts, Naylor, Davis, Jones, Beeson et al., 2006).

In large scale epidemiological studies, Body Mass Index (BMI) or the Quételet Index is commonly calculated from a participant's weight and height (kg / m^2). This is cheap to do and straightforward to calculate. The index has the same dimensions as density and pressure would in a 2D world. Adolphe Quételet himself realized that these dimensions ideally should vary for different age groups, with babies' index being scaled by 3 (cubed- as they are more rounded: now known as the Ponderal Index), adolescents' index being scaled by 2 (squared- as they tend to be thinner) and adults' index being scaled by 2.5 (increasing from adolescents' as they gain weight as they age). Nevertheless, a study comparing the Ponderal Index with BMI using DXA showed no significant improvement in body fat estimation using a cubed formula rather than squared (Tovée, 2012). Despite these criticisms, BMI has provided a robust proxy for percentage body fat (Romero-Corral, Somers, Sierra-Johnson, Thomas, Collazo-Clavell et al., 2008), and is more reliable than BIA and SFT (Chan, Leung, Lam, Peng & Metreweli, 1998).

Other simple and cheaply available methods to estimate body fat are the waist-to-hip ratio (WHR), waist circumference (WC) and waist-to-height ratio (WHtR). Firstly, WHR has been used as an alternative to BMI and many have suggested it is a better measure of body fat as it takes into account the shape of the body instead of just its size (Singh & Singh, 2011). WHR is calculated by measuring the subject's waist and hip circumferences and dividing the waist measure by the hip. Measures of the waist are taken at the smallest part of the torso/natural waist or if the

waist is convex (as in pregnancy), the measure may be taken 1 inch above the line of the navel. Hip circumferences are measured at the widest part of the buttocks or hips. Research has shown that estimates of adiposity from WHR are confounded by the fact that WHR is highly age-dependent (Ketel, Volman, Seidell, Stehouwer, Twisk et al., 2007) and it is relatively unreliable in extreme body shapes, such as in the obese and emaciated (Ketel et al., 2007). Furthermore, it has been suggested that individual differences in the proportions of participants' skeletons may confound WHR (Ley, Lees & Stevenson, 1992). Finally, WHR does not account for participants' height which can, for mechanical reasons, affect bone thickness in the pelvis and thighs. In comparison, waist circumference (WC) has been reported as a better measure of adiposity than WHR (Taylor, Keil, Gold, Williams & Goulding, 1998) and this is particularly true for visceral fat (Rankinen, Kim, Perusse, Despres & Bouchard, 1999). WC is often combined with a height measurement (waist circumference-to-height ratio), therefore taking into account overall size as well as shape, and unlike WHR, WHtR is also said to not be dependent on age (Aeberli, Gut-Knabenhans, Kusche-Ammann, Molinari & Zimmermann, 2011).

Owing to their relative ease of measurement, we have chosen to use BMI and skin fold thickness to estimate adiposity for Experiment 3, Chapter 4. We chose not to use WHR for the reasons outlined above.

1.7. Comparing BID in eating disordered participants and controls

In an early comparison of body image distortion in patients with anorexia and controls, Slade and Russell (1973) asked participants to estimate body part widths across the face, chest, waist and hips, and they compared these estimates to actual

measurements obtained by anthropometer. Body part estimates were obtained using an apparatus which required participants to judge when two horizontally aligned lights had been adjusted, by the experimenter, to match the width they believed the body part to have. To test for non-specific perceptual distortion, the authors also asked the AN patients to carry out a tactile size judgement task on wooden blocks. Slade and Russell (1973) found that patients with AN (who were on average 20kgs lighter than the controls) did overestimate their body size for all four sites, whereas the control participants were very accurate in their estimations. Importantly, the AN patients estimated the block sizes accurately, and performance in this control task was uncorrelated with performance in the body size estimation task, suggesting that the perceptual distortion was specific to their body parts. In a second experiment, AN participants were asked to judge the widths of the same four body parts, and the height of themselves as well as another, a model of another person. The authors replicated the over-estimation of the width of the four body parts for estimations of self, and found that this extended also to estimating others. However, no inaccuracies were found for height measurements of either self or the model. While there is some evidence suggesting that women with AN may under-estimate their body size (Meerman, 1983), or show performance in size estimation tasks equivalent to non eating-disordered controls (Fernández, Probst, Meerman, & Vandereycken, 1994; Probst, Vandereycken, Van Coppenolle & Pieters, 1995), most studies concur with Slade and Russell (1973) and have found that patients with AN overestimate their body size (Gardner & Bokenkamp, 1996; Gardner & Brown, 2014; Tovée et al., 2003).

However, as Slade and Russell (1973) point out themselves: “ .. *the term 'perceptual distortion' has been freely used to refer to the systematic size estimation*

errors observed. However, the error tendency might equally reflect a judgmental as opposed to a purely perceptual process. This seems unlikely in view of the demonstrated specificity of the errors made but the possibility should be borne in mind". This is a critical point, and returns to the central distinction between an attitudinal versus a perceptual explanation (or both) for why AN participants overestimate body size. Essentially the same point is made by Smeets et al. (1999): "... *Two explanations will be considered. According to the first, a disturbance of body image implies a disturbance of visual perception. The body is imaged as fatter because it was originally perceived as fatter. Here, the assumption ... is that imagery is equivalent to retrieving a previously perceived visual pattern from visual memory (a bottom-up approach). According to the second explanation, the disturbance occurs at the stage of imagery. Because she thinks she is fat, the individual with AN (most often a female) constructs a visual image of herself as fat (a top-down approach)*".

In principle, Signal Detection Theory (Green & Swets, 1966) offers a way to distinguish between these possibilities unambiguously. Put succinctly, an observer's ability to perform a detection/discrimination task is limited by internal noise. The decision that an observer makes on any trial of such a task – e.g., is a stimulus present, yes or no, is driven by two factors: (i) the information they have (e.g., signal strength) and (ii) the criterion or internal bias that an individual sets for making a decision. Because there are two components (signal strength and criterion) determining the outcome of each trial in the task, two measurements are needed to characterize the task performance. In the context of, for example, a two alternative forced-choice (2AFC) task, by measuring both hits and false alarms, it is possible to estimate both d' , which is a measure of an observer's task sensitivity, independent of

their criterion, as well as an estimate of the criterion, or bias itself (β). Indeed Smeets et al. (1999) carried out such an experiment by asking AN patients and healthy controls to judge pairs of images using the method of constant stimuli. On each trial of the critical experiment, participants saw two images of a body, side by side. One image, the reference, was an image of themselves, and the other was an image of themselves which was compressed/expanded in the horizontal dimension to reflect a range of body widths. Participants were asked to judge whether the pair of images was same or different. Smeets et al. (1999) applied signal detection analyses which preserved the directions of the stimulus size changes (i.e. thinner or fatter), and showed that AN patients had a significant *bias* for responding “thinner than”, even though they were just as sensitive to the task as controls. This study is one of the only three that we are aware of in which signal detection theory has been applied to the perception of body size in anorexia. However, while valuable, the Smeets et al. (1999) study fails to address the question of primary interest which is asking the participant to tell “what size do I believe I am?”. To do this, the authors would have had to be able to manipulate the signal of interest (the AN participants’ belief), which by definition they could not do, because both the signal and the observer bias “reside” in the mind of the observer, and cannot be accessed directly. Instead the Smeets et al. (1999) study seems to address the question “how sensitive am I to telling apart those two pictures of me?”. Signal detection theory can be applied here because the investigators have control over the signal in the experiment – i.e. the image that is displayed on the PC monitor, and, arguably, the participant can therefore solve the task without having to refer to their internal memory/representation for their own body shape/size. Given this impasse, we turn therefore to studies which have used the method of constant stimuli and applied

classical psychophysical methods to measure: (i) the point of subjective equality (PSE) which corresponds to the body size that *participants believe themselves to have*; (ii) the difference limen (DL) which corresponds to how sensitive a participant is to changes in body size. The problem with these methods is that both PSE and DL are influenced by the subjective states of the observer – for example their expectancies about the stimuli (Gescheider, 1997). Nevertheless, it is possible to see dissociations between the two measures, which as we will see are useful and interpretable. Moreover, Gardner, Jones and Bokenkamp (1994) did carry out one study in which they compared performance in the same participants on body size estimates obtained using the method of limits, the method of constant stimuli and a signal detection approach. It is reassuring that these authors found a significant correlation between PSE from the method of constant stimuli and β (i.e. bias) from the signal detection approach. With regard to sensitivity, they also found a correlation between DL and d' , although this was not significant.

Gardner and Bokenkamp (1996) used the video distorting technique (VDT) to measure the smallest horizontal distortion to body width that 35 AN and BN female participants and 19 healthy female controls could detect. Each participant was photographed and had their pictures modified according to the VDT. The authors then applied adaptive probit estimation (Watt & Andrews, 1981) to the method of constant stimuli in order to estimate both the DL and PSE for each participant. They found significantly larger PSEs on average for AN participants than for controls, but no difference between the groups for DL. These results are entirely consistent with participants with AN believing themselves to be larger than they actually are, even though they were just as sensitive in the psychophysical task as controls. Similar findings using the same psychophysical procedures with either the same or very

similar stimuli were reported by Gardner and Moncrieff (1988) and Mussap, McCabe and Ricciardelli (2008). The fact that these results to some extent mirror Smeets et al. (1999) suggests that choosing a classical psychophysics approach over signal detection may not be such a poor compromise.

1.8. Contraction bias and the role of body mass index (BMI)

Even if classical psychophysics reveals a reliable difference in PSE between AN participants and controls, as Cornelissen et al. (2013) point out, this could come about in one of two ways. Firstly there might be a pathological process specific to individuals with AN and a non-pathological process found in non-eating disordered individuals which modulate the relationship between body size estimation and actual BMI in different ways. These possibilities are illustrated in Figure 1.5 by the “single channel” and “dual channel” models, overleaf. In both models, observers’ estimates of their body size, expressed in BMI units, are plotted as a function of observers’ actual BMI. In the “dual channel” model, the body size over-estimation by observers with AN can be explained if the intercept for the regression of estimated BMI on actual BMI for this group was sufficiently higher than that for controls. As a result, values for estimated BMI (i.e. PSE) would be drawn from two separate distributions with a significantly higher mean for AN observers than controls. However, exactly the same difference between means could be generated by an alternative, “single channel” model. Here, all that is required is a single distribution of individual differences in the performance of one psychophysical process, common to the general population.

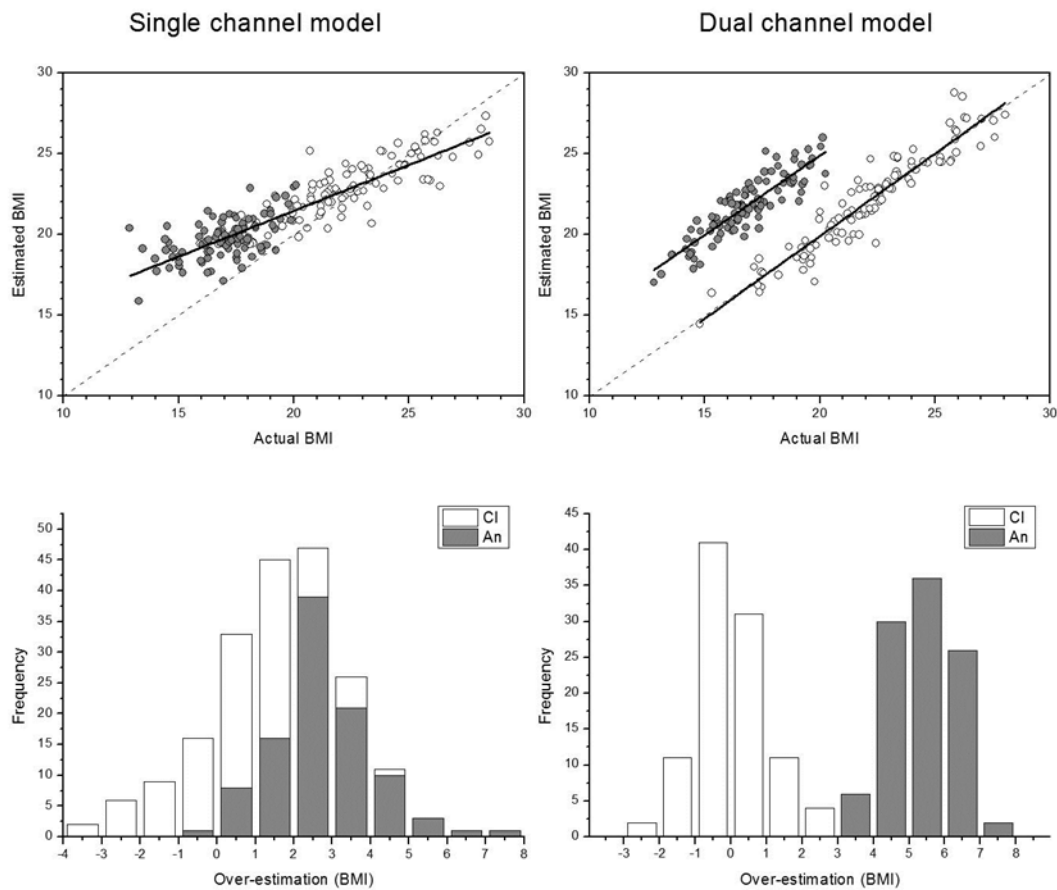


Figure 1.5. Sketch graphs to illustrate the single and dual channel models. Top row: scatterplots of estimated BMI as a function of observers' actual BMI (Anorexics = filled dots; Controls = empty dots). Dotted lines represent correct judgements. Bottom row: distributions of participants' over- / under-estimation of their body size for each model. Note over-estimation = estimated BMI – actual BMI. White bars, non-eating disordered controls; gray bars, anorexic observers.

Differences in mean body size over-estimation between individuals who do or do not have AN would then depend only on whether individuals with AN are over-represented in one part of that distribution. The critical difference between the two models is that the psychophysical behaviour of observers who have AN does not need to be pathological. For example, a well established bias in magnitude estimation, known as contraction bias (Poulton, 1989), could produce this effect.

Contraction biases “*can affect any kind of quantitative judgement or rating. In the absolute version of the stimulus contraction bias, magnitudes larger than the observer’s reference magnitude are underestimated. Magnitudes smaller than the observer’s reference magnitude are over-estimated*” (Poulton, 1989). Humans learn a reference body size / body weight which, for each individual, is based on the sample of bodies they have seen over the course of their life. In a large sample of observers, this will approximate to the population average. It is also proposed that this reference is weighted towards more recent experience (e.g., Leopold et al., 2001; Rhodes, Jeffery, Boeing & Calder, 2013; Winkler & Rhodes, 2005). Contraction bias therefore predicts that people will be most accurate at judging body sizes closest to the reference and increasingly less accurate as the size of the body being judged and that of the reference diverge. Bodies larger than the reference will be increasingly underestimated and bodies smaller than the reference increasingly overestimated. Quantitatively, sufficient evidence to suggest the presence of contraction bias can be derived from a plot of estimated weight (y-axis) as a function of stimulus weight (x-axis). We should see: (i) a slope for the regression of estimated weight on actual weight which is statistically significantly less than 1 – i.e. less than the slope of the line of equivalence where estimated weight agrees perfectly with actual weight; (ii) that this regression line crosses the line of equivalence approximately at the mean weight of the reference population.

Returning to the single channel model in Figure 1.5, people who have AN usually weigh less than those who do not have AN. Therefore, the estimated BMIs of observers who have AN will necessarily be drawn from that part of the distribution where there is greater overestimation in general. Consequently, this sampling bias alone would be sufficient to produce a significant difference between the means of

the observer groups. To distinguish between these two models, Cornelissen et al. (2013) re-analysed data from Tovée et al. (2003) and found evidence entirely consistent with the “single channel” model. Moreover, in a second prospective experiment, Cornelissen et al. (2013) used the VDT to explore the relationship between PSE, DL and a number of the psychometric tasks typically used to assess ‘attitudinal’ aspects of body image, such concern about body shape and weight, tendency towards depression and self-esteem. These authors showed clear relationships between PSE and the psychometric dimensions but not for DL. Together, these results suggest that a fuller understanding of body image distortion will need to take account of *observer* BMI, because of perceptual effects like contraction bias, and, simultaneously a contribution from the psychometric dimension in parallel – the subject of Experiment 3 in Chapter 4.

1.9. Non-body size estimations in eating disordered participants and controls

A number of studies have attempted to investigate purely visual and visuo-tactile disturbance in AN in an attempt to explain body size overestimation. Most of these have shown that AN participants estimate the size of non-body objects accurately (e.g., Garner et al., 1976; Slade & Russell, 1973; Urgesi, Fornasari, Perini, et al., 2012). However, a small number of studies have shown intriguing differences between participants with AN and healthy controls. For example, Case, Wilson and Ramachandran (2012) showed that the size-weight illusion was dramatically reduced in participants with AN. Normally, when two objects of equal weight but different sizes are held, people perceive that the smaller object feels much heavier than the larger object because of an implicit assumption that weight scales

with size (see also Madsen, Bohon & Feusner, 2013). There is also a group of studies suggesting that AN individuals may have low level visual sensory deficits. For example, Lawrence, Dowson and Foxall (2003) showed that AN participants were impaired in a dynamic categorization task equivalent to one known to activate dopamine neurons in primates, and sensitive to dopaminergic manipulations in humans. Moschos, Gonidakis, Varsou, Markopoulos, Rouvas et al. (2011) showed that AN patients suffered a decrease in macular and retinal nerve fibre layer thickness, as well as a decrease in the electrical activity of the macula. These changes were not associated with any measureable change in low level visual sensitivities. Since most of these studies have investigated individuals with very low BMI, it is likely that at least some of these effects might be attributable to starvation and its attendant impairments of cognitive function.

1.10. Treatment methods for eating disorders

The primary aim of most treatments for eating disordered patients is first to restore an individual's weight to a healthy body mass index (BMI), using a combination of medical, nutritional and psychological methods. Whilst drug treatments are still widely used for bulimia nervosa in particular (Shapiro et al., 2007), in recent years, more emphasis has been placed on psychological treatment methods: ameliorating body size and shape concerns, pathological eating patterns, and dealing with precipitating psychological and social factors (American Psychiatric Association, 2006). Unfortunately, even though a variety of psychological treatment methods have been tried, there are very few randomised controlled trials in this field. Reviews of efficacy have failed to identify substantial

differences in outcome between different psychological treatment approaches. Nevertheless, current practice probably favours a multidisciplinary approach over a single, unified approach to treatment (American Psychiatric Association, 2006; Hay, Claudino, Touyz & Abd Elbaky, 2015; Watson & Bulik, 2013).

Historically, psychodynamic approaches have been used, and these derive from Bruch (1962) establishing the link between disturbances in the psychology of an individual who has anorexia and their disordered eating behaviour. Psychoanalytic approaches have also been attempted (e.g., Dare, 1995), where the conscious and unconscious meanings attributed to eating disordered symptoms are explored. Focal Psychodynamic Therapy focuses on interpersonal relationships (Zipfel, Wild, Groß, Friederich, Teufel et al., 2014), whereas Cognitive Analytic Therapy (CAT) combines Cognitive Therapy and Focused Psychodynamic Therapy to explore eating disorders as an expression of self within interpersonal relationships (Dare, Eisler, Russell, Treasure & Dedge, 2001). In addition, Feminist Therapy (Striegel-Moore, 1995), Motivational Enhancement Therapy (Treasure, Todd, Brolly, Tiller, Nehmed et al., 1995), the Maudsley Model (Schmidt, Oldershaw, Ichi, Sternheim, Startup et al., 2012), the Specialist Supportive Clinical Management Approach (McIntosh, Jordan, Luty, Carter, McKenzie, Bulik et al., 2006) and the Karolinski Madometer Treatment (Bergh, Brodin, Lindberg & Sodersten, 2002) have all been tried and claim variable success in terms of positive outcomes for patients. Interpersonal Psychological Therapy (IPT) has shown some good outcomes for bulimia nervosa (Fairburn, Jones, Peveler, Carr, Solomon et al., 1991), and family-based therapy for anorexia nervosa (Fairburn et al., 2003). Cognitive Behavioural Therapy (CBT) (Fairburn et al., 2003; Garner, Vitousek & Pike, 1997) has been widely explored in the area of eating disorders, and a relatively

good outcome has been identified for bulimia nervosa, but not so much for anorexia nervosa (Butler, Chapman, Forman & Beck, 2006; Channon, De Silva, Hemsley & Perkins, 1989; Fairburn et al., 2003).

Psychotherapy (of some form) is usually applied once the primary aim of medical stabilisation is well under way. However, it is not uncommon for psychotherapy to be concluded, sometimes at the insistence of the patient, once an individual is showing good weight gain. (see e.g., Halmi, Agras, Crow, Mitchell, Wilson et al., 2005). In those controlled trials that do exist, the focus has usually been on body weight (or BMI) gain (see, e.g., Hay et al., 2015), which does not necessarily reflect psychological recovery. In fact, body image distortion often persists and can even worsen temporarily following body weight restoration (e.g., Deter & Herzog, 1994; Goldbloom & Olmsted, 1993; Windauer, Lennerts, Talbot, Touyz & Beumont, 1993). Furthermore, even when improvements are seen in behaviours like eating, dieting and purging, body dissatisfaction scores may remain stubbornly high (Davis, Fox, Cowles, Hastings & Schwass, 1990). At the same time, persistent body image distortion has been shown to predict relapse in eating disorders (e.g., Button, Fransella & Slade, 1977; Carter et al., 2004; Channon and De Silva, 1985; Freeman, Beach, Davis & Solyom, 1985; Slade & Russell, 1973). Consequently, if body image distortion is not dealt with adequately before a weight restored patient is discharged from inpatient care, they may already be carrying the seeds of their own relapse.

1.10.1. Specific therapies for Body Image Distortion (BID)

There are a number of treatment methods specifically targeting distorted body image. For example, burning newspaper images of thin people, writing letters

to body parts, body massage and controlled body exposure (see e.g., Fairburn, Cooper & Cooper, 1986). However, many of these methods lack a strong theoretical rationale, and are loosely based on the assumption that socio-cultural influences have caused the eating disorder. Again, there is a relative lack of existence of randomised controlled trials.

Interestingly, there is evidence that some pharmacological approaches can reduce body image dissatisfaction (e.g., Goldbloom & Olmsted, 1993). However, it is unclear whether these improvements in body satisfaction are drug specific effects or whether they are tied to non-specific improvement in the eating disorders in general. Cognitive Behaviour Therapy (CBT) has also been combined with a mirror exposure desensitization approach in anorexia nervosa, with the aim of encouraging patients to pay less attention to changes in body shape and weight, and therefore to reduce the salience of cues that would otherwise fuel body image distortion (e.g., Key, George, Beattie, Stammers, Lacey & Waller, 2002). This approach resulted in sustained reductions in body image dissatisfaction, although the sample consisted of only six individuals. More recently virtual reality (VR) techniques have been tried (Marcoa, Perpiñá & Botellac, 2013; Riva, Bacchetta, Baruffi & Molinari, 2002), but again only with very small participant numbers and are therefore inconclusive to date.

1.11 Research questions

The weight of evidence presented in the Introduction suggests that women with AN do indeed over-estimate their body-size, on average, when compared to healthy controls. However, questions remain about what are the mechanisms that

drive the perceptual contribution to body image distortion, and how this is related to the attitudinal component. The primary aims of the research reported in this thesis are therefore to obtain a coherent account of body image distortion in AN by measuring, in the same individuals: (i) the perceptual responses to body size estimation using the most ecologically valid stimuli we can build together with robust psychophysical methods, (ii) attitudinal influences on body size estimation using a battery of well validated, normed psychometric tools, (iii) taking account of how the BMI of the observer influences body size estimation. We will then model statistically the inter-relationships between these three domains in participants with AN and in healthy controls. Having obtained such a description, we will then test a novel approach to the specific treatment of body image distortion.

Chapter 2

Methods

2.1. Computer Generated Imagery (GCI) for stimulus creation

For the reasons outlined in the Introduction, the experiments in this thesis used the same computer-generated imagery (CGI) methods as the film and games industries to create 3D images representing a full spread of BMI. This strategy therefore amounts to an updated version of a figural rating scale, like the Stunkard scale (Stunkard, Sorensen & Schulsinger, 1982), with the advantage of a continuous variation in BMI, as well as highly realistic 3D imagery. The disadvantage, of course, is that it requires individual participants to relate their internal body shape representation to variation in one particular individual. For a more detailed appraisal of the limitations of this methodology see section 8.4.2. (pages 182-185).

All the CGI stimuli were created in the Daz Studio v4.8 modelling environment. This program allows subtle manipulation of the body shape and posture of a fully rigged digital model. We used the Victoria 5 and 6 character models, which are based on the Genesis 1 and Genesis 2 female base models respectively, in Daz Studio. From the neck down, there are 320 body shape controls, 16 of which influence whole body attributes such as adiposity. From the neck up there are 209 controls for head shape. For experiments 2, 3, 5, 6, and 7, we modified the Victoria 5 and 6 character models to capture the average body shape of a 25 year old UK Caucasian female, and this provided our baseline models whose adiposity we could then vary systematically. To do this, we extracted the appropriate averages from the Health Survey for England (HSE) 2008 dataset to select the model's height, leg length, bust circumference, under-bust circumference, waist circumference and

hip circumference. In addition, we ensured that these baseline models had an average 25-year old female's torso-to-leg ratio and waist-to-hip ratio.

The first question was whether participants judged the Victoria 5 and 6 baseline models to be plausible representations of female body shape. To address this question, we applied the adiposity morphs to render a set of three images intended to capture the underweight, normal weight and overweight classifications defined by the World Health Organization (WHO). We then asked 30 participants who were recruited from amongst friends and colleagues to provide qualitative feedback about these images. In addition, we carried out two further comparisons. First, the 3D volumes of the CGI modelled bodies were compared to a 3D statistical model of the relationship between BMI and shape changes in 114 scanned bodies (Hasler, Stoll, Sunkel, Rosenhahn & Seidel, 2009). Secondly, we compared our models qualitatively to digital photographs of 220 women in a standard pose who vary in BMI from 11 (emaciated) to 45 (obese) (Tovée, Maisey, Emery & Cornelissen, 1999). Based upon all the feedback we received, we further modified our baseline model by reducing chest size and shape to represent a more naturalistic breast shape, made the lips thinner, the eyes smaller and cheeks (buccae) flatter.

For experiment 4, where we needed to create an individual avatar of each participant based on their 3D body shape scan, we used a slightly different modelling strategy. For this experiment, each participant was first scanned, using a Size Stream Body Scanner, and the data converted to a 30k polygon mesh. This is a smooth surface shell that can be read into Daz Studio to provide a template to which the Genesis 2 female baseline model could be fitted (rather than manipulating either of the Victoria character models). From a modelling point of view this made sense because the Genesis baseline models are deliberately designed to provide maximum

shape variability. For further details of avatar model fitting, see section 5.2.3. (pages 115-116). One limitation with this technique however, is that the resolution of the Size Stream 3D body scan is not sufficient to capture fine surface details of body muscularity. For example, while distinctions in rectus abdominis divisions on the abdominal wall can be seen, if present, individual forearm muscles cannot. Therefore, avatar muscularity was to some extent adjusted subjectively. This was possible because avatars were created as soon as possible after a participant's body scan had been captured and she had left the laboratory.

2.1.1. Calibrating model BMI

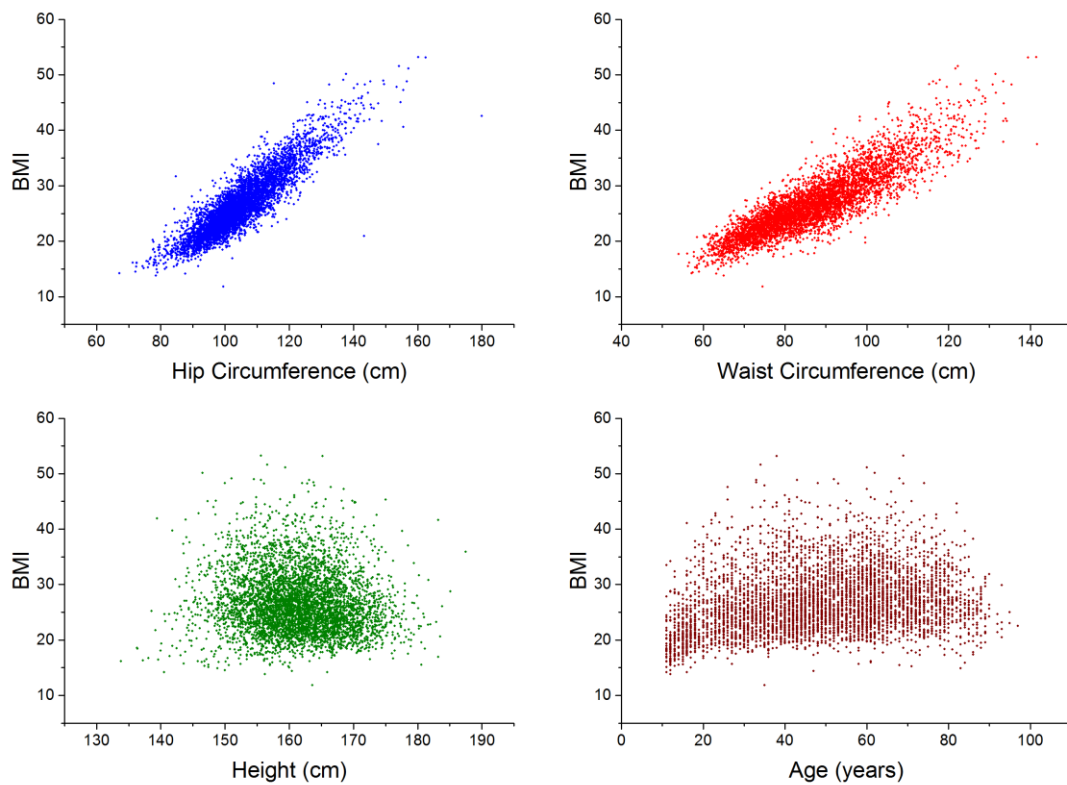


Figure 2.1. Scatter plots of BMI as a function of the hip and waist circumferences, height and age of the 4976 Caucasian females in the HSE 2008 dataset.

Ultimately, we wanted to use our CGI models to represent the range of body shape changes over a wide BMI range, and for these stimuli to be accurately calibrated for BMI. To do this we first needed to derive a calibration equation which related model waist and hip circumferences, height and age to BMI. Therefore we used the Health Survey for England (HSE) 2008 dataset which contains information about BMI, height, waist circumference, hip circumference and age from 4976 Caucasian females in the UK. Figure 2.1 above shows scatterplots of the relationships between BMI and waist, hip, height and age for these individuals. To derive the calibration curve we used PROC REG in SAS v9.3 to compute a multiple regression of BMI on waist, hip, height and age from the HSE 2008 dataset. Based on inspection of Figure 2.1 we permitted second order polynomial fits for height and age, and optimized the model fitting by maximizing r-square and minimizing Mallow's CP. The best fit model explained 90.24% of the variance in BMI and was:

$$y_i = \beta_0 + \beta_1 \cdot x_1 + \beta_2 \cdot x_2 + \beta_3 \cdot x_3 + \beta_4 \cdot x_4 + \beta_5 \cdot x_4^2 + \epsilon_i$$

Where y_i = BMI, x_1 = hip circumference, x_2 = waist circumference, x_3 = height, x_4 = age, $\beta_0 = 9.676$, $\beta_1 = 0.308$, $\beta_2 = 0.150$, $\beta_3 = -0.179$, $\beta_4 = 0.0554$, $\beta_5 = -0.000762$. Each term in the model was statistically significant at $p < .0001$.

Within the Daz Studio modelling environment, the height of our model was adjusted to 1.65 m, this being the average height for females aged 18 to 45 in the UK (HSE 2008). Because our model exists in a 3D world which can be scaled 1:1 with the real world, the model's waist and hip circumferences can be measured and BMI estimated from the calibration equation above. To extract these measurements, we used the Measure Metrics tools in Daz Studio. To do this, we first used the animation tools in Daz Studio to set a number of key frames for three of the whole body

controls for adiposity. The controls are called: ‘emaciated’, ‘thin’ and ‘heavy’ and each controller varies the adiposity shape morph it is responsible for between a minimum of 0 and a maximum of 1. Therefore, to generate a sequence of animation frames from the thinnest to the fattest body that the Genesis models can represent, we used the following key frame settings:

Key Frame 1: emaciated = 1, thin = 1, heavy = 0

Key Frame 2: emaciated = 0, thin = 1, heavy = 0

Key Frame 3: emaciated = 0, thin = 0, heavy = 0

Key Frame 4: emaciated = 0, thin = 0, heavy = 1

The next question was how many animation frames should we allow Daz to interpolate between these key frames. Pilot testing showed that by allowing DAZ to animate the key frame sequence over 310 frames would allow us to pick a subset of 120 images, covering the BMI range 12 to 42.5 in 0.25 BMI increments, with an accuracy of +/- 0.07 BMI units per frame. Moreover, because the size of the BMI change between key frames from 3 to 4 was approximately twice that for either key frames 1 to 2 or from 2 to 3, we needed to assign approximately twice the number of animation frames to that sub-sequence in order to achieve the same accuracy in BMI increments. This is reflected in the larger gap between key frame markers 3 & 4, than for the markers between 1 & 2 and 2 & 3 in Figure 2.2 overleaf.

In practice, we used the Measure Metrics tool to extract waist and hip measurements from every 10th frame in the 310 frame sequence. For the animation frames in between the measured frames, we assumed linear increases in waist and hip circumferences and interpolated these values accordingly. Figure 2.2 overleaf shows a plot of the relationship between model waist and hip circumferences as a function of animation frame number. We then applied the waist and hip

measurements to the calibration equation above to identify which of the 310 animation frames corresponded closest to the BMI range 12.5 to 42.5 in 120 increments of 0.25 BMI units, and rendered only those images for our stimulus set at 1024h x 576w pixels (24bit colour depth). This also meant that the underlying skeletal proportions, posture, skin, clothes, hair and lighting did not change through the stimulus sequence – only adiposity.

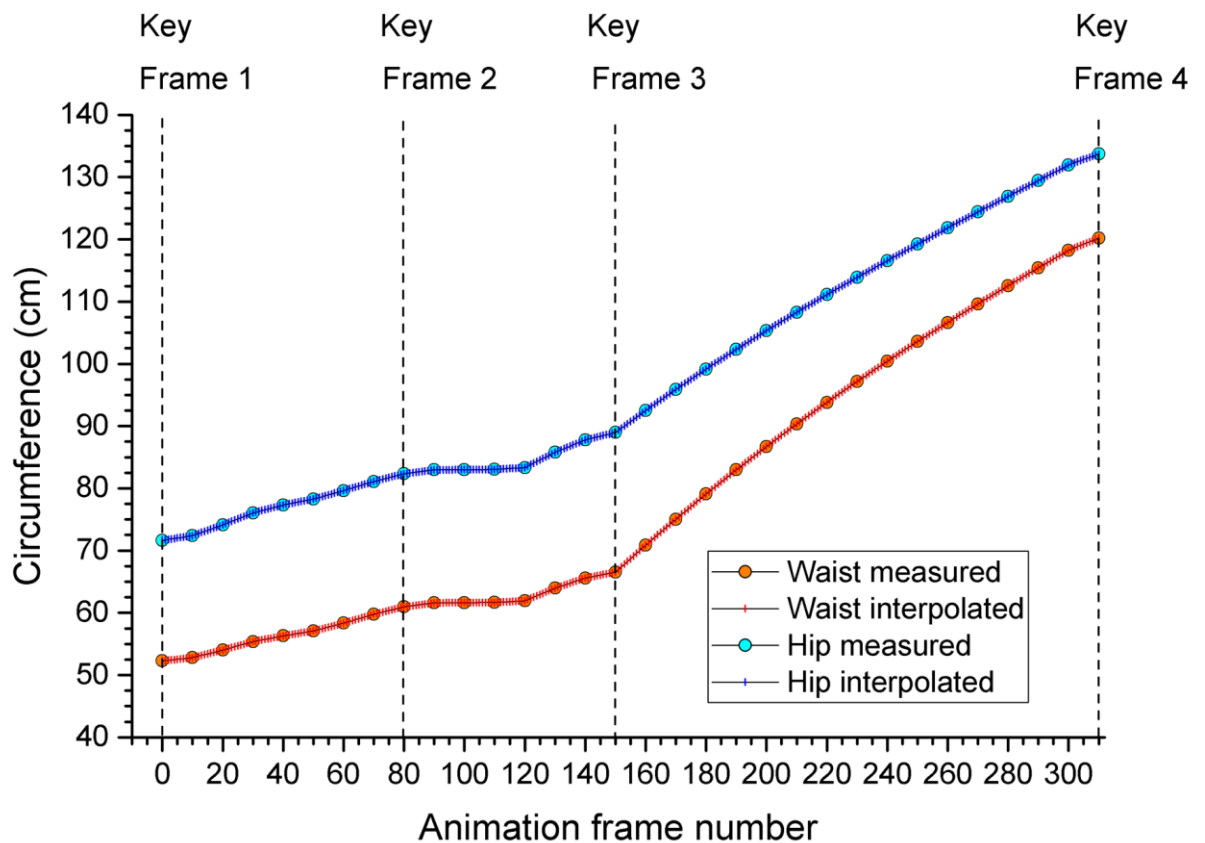


Figure 2.2. A plot of CGI model waist and hip circumference as a function of animation frame number, together with the location of the animation Key Frames (see text). We used PROC EXPAND in SAS v9.3 to linearly interpolate the 9 values between each measurement point.

Figure 2.3 below shows three representative images from the stimulus database.

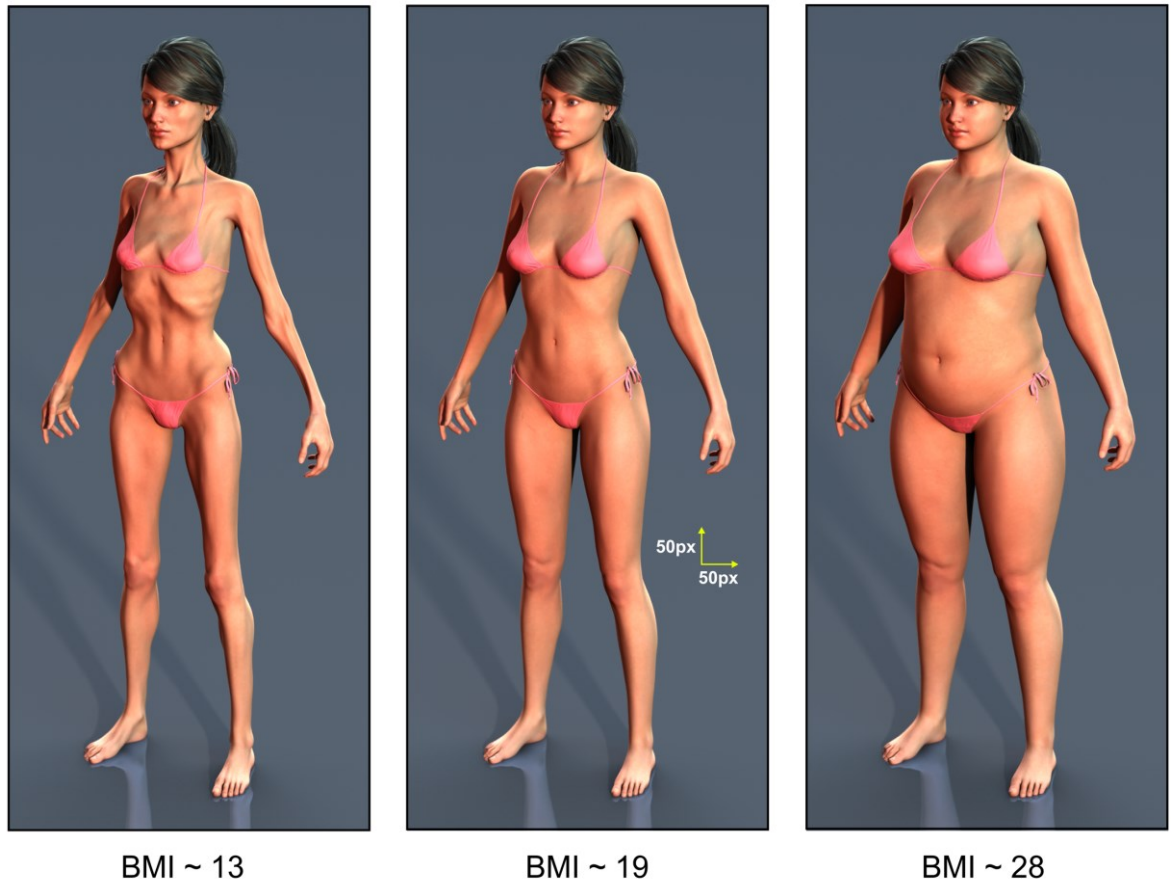


Figure 2.3. Representative 3D rendered stimuli at three different BMI levels.

2.2. Classical psychophysical methods

The data for the studies were collected either by using the method of limits approach where an individual needs to make their choice by adjusting a slider provided on the computer screen (Experiments 1 & 4), or using the method of constant stimuli, configured as a 2-alternative forced choice (2AFC) discrimination

paradigm (Experiments 2, 3, & 7) (Gescheider, 1997). Programmes to run these classical psychophysics methods were written in Python by Andre Bester.

2.2.1. Method of constant stimuli

The method of constant stimuli was implemented as a two alternative forced choice task (2AFC) to measure: (i) the point of subjective equality (PSE) which is defined from the psychometric function as the BMI at which participants respond 'larger' 50% of the time. This value corresponds to the body size that participants believe themselves to have; (ii) the difference limen (DL) is the amount of change in a stimulus required to produce a just noticeable difference – in this case between the participant's image of self and the image on screen. The DL has a lower and an upper part. The lower part is the difference in BMI falling between the 25% 'larger' response points on the psychometric function and the PSE. The upper part is the difference in BMI falling between the 75% 'larger' response points and the PSE. As is commonly the case, we averaged the lower and upper part to give a single estimate of DL. The DL captures the steepness of the psychometric curve and corresponds to how sensitive a participant is to changes in body size (see Figure 2.4).

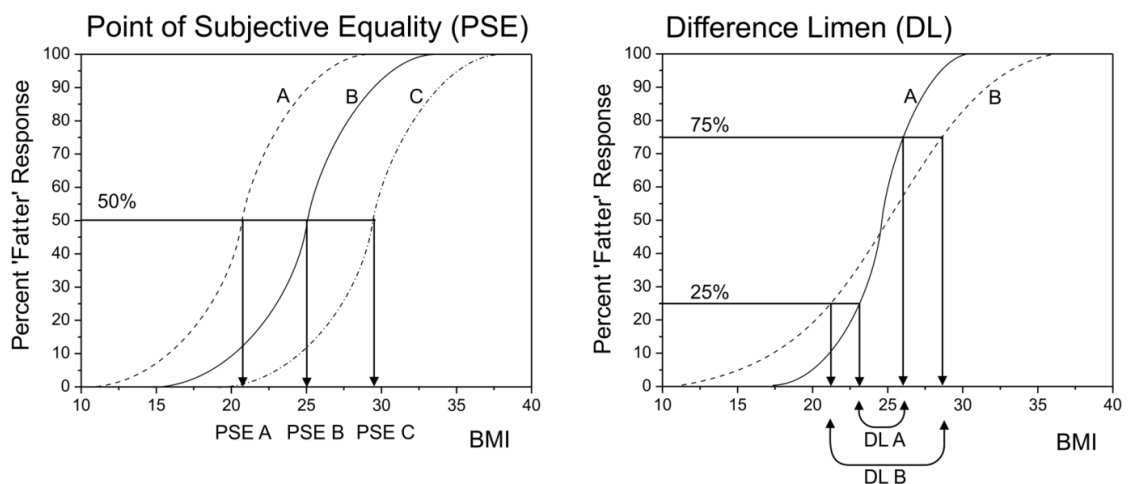


Figure 2.4. A graphical illustration of how the psychometric function for body size estimation can be used to separate out sensory sensitivity (indexed by the difference limen, DL) from perceptual bias (indexed by the point of subjective equality, PSE). On the left, participants A, B & C might all have the same BMI of 25. However, participant A under-estimates and participant C over-estimates their body size. On the right, participant A is more sensitive to body size change than participant B, and therefore has a steeper psychometric function, with a smaller DL.

In the 2-AFC task, participants were presented with a randomized sequence of CGI images of the standard model. Across the image set, BMI varied continuously from 12.5 to 42.5. On each trial of the task, one image was presented and participants were required to decide, and respond by keyboard button press, whether the body depicted was larger or smaller than themselves. Stimuli were presented on a 19" flat panel LCD screen (1280w x 1024h pixel native resolution, 32-bit colour depth) for as long as it took participants to make a decision. At the standard viewing distance of ~60cm, the image frame containing the CGI model subtended ~26° vertically and ~8° degrees horizontally. To take full advantage of the method and at the same time reduce participant time involvement, the 2AFC task made a rough estimate of PSE, followed by more detailed testing. For the first two blocks, 9 stimuli were used which covered the full BMI range from 12.5 to 44.5 in equal steps, and presented each stimulus 10 times. At the end of the two blocks, a cumulative Gaussian curve was automatically fitted to each dataset to estimate the PSE, and the BMI corresponding to the mean of these two estimates was calculated. For the final block, based on extensive pilot testing, we used 21 stimulus images which were selected to have a BMI range centred on the mean defined by the first

two blocks, and covering ± 2 BMI points in 0.25 BMI steps. Each image was presented 10 times at the end of which a cumulative Gaussian curve was fitted in order to give the definitive estimate of PSE and DL.

2.2.2 Method of limits

For our avatar stimulus sequences in Experiment 4, participants used the method of limits to estimate their body size under each of four task conditions whose order was randomized across participants. Participants had to decide: the size and shape they believed themselves to have while viewing the whole avatar (WHOLE); the size and shape they believed themselves to have when they could only see the avatar from the waist down to the legs (LEGS); the size and shape they believed themselves to have when they could only see the avatar from the waist up to the head (TORSO); the size and shape they would ideally like to have when viewing the whole avatar (IDEAL). For each condition, participants carried out 20 trials using the same display setup as for the 2AFC task. On each of these 20 trials, the participant's avatar appeared on screen with the face blurred. Beneath the avatar was a slider control. The participant was asked to click on the slider control to move it from side to side. When the slider moved leftwards the BMI of the avatar reduced smoothly to a minimum of ~ 13 and increased to a maximum of ~ 45 when the slider moved rightward. The participant had to decide which image in this continuum fitted best the criterion for the particular experimental condition, and then press a radio button on screen which allowed the stimulus PC to log their response and initiate the next trial. At the start of each trial, the BMI of the avatar was set randomly to either its minimum, with the slider appearing at the leftmost extreme of its range of movement, or the maximum BMI, with the slider appearing at the rightmost extreme

of its range of movement. Figure 2.5 below illustrates four sequential frames from the MOL task.



Figure 2.5. Body shape changes for an anonymized, bespoke avatar in Experiment 4, as the slider control is moved from left to right through screenshots A, B, C & D.

2.3. Psychometric measures

We used a number of validated self-report questionnaires to investigate individuals' attitudes towards their body weight and shape, their attitudes to eating,

their tendency towards depression and their self-esteem. Participants were instructed not to fill in all the questions in the questionnaires in the event that doing so was causing them distress. Furthermore, the questionnaires were not examined by the researcher immediately after the participants completed them. This was to ensure that not only was any embarrassment to the participant avoided, but also to encourage the participants to be as truthful as possible.

To investigate the body shape and size concerns and eating habits three main questionnaires were used: The Eating Disorders Examination Questionnaire (EDE-Q), The Eating Attitudes Test (EAT), and the 16-item Body Shape Questionnaire (BSQ 16b).

The EDE-Q (range 0-6) is a self-report version of the Eating Disorder Examination (EDE) structured interview (Fairburn & Beglin, 1994). This questionnaire measures independently four different aspects of eating disordered behaviour, two of which are directly related to body image distortion, and therefore particularly relevant to the investigations in this thesis. In addition, it is possible to calculate a global eating disorder score by compiling the responses across all four dimensions, and this is used clinically to identify individuals who might have eating disorders. The questionnaire is also designed to obtain frequency data on some eating disorder behaviours.

The four dimensions of the EDE-Q are each measured using from five to eight questions for which responses are required on a 6-point Likert Scale. First, the restrictive nature of an individual's eating behaviour is assessed using the Restraint Scale (EDE-Q res). The eating concern scale (EDE-Q eat) measures preoccupation with food and social eating; the Shape Concern subscale (EDE-Q sc) measures

dissatisfaction with one's own body shape and the Weight Concern subscale (EDE-Q wc) measures dissatisfaction with body weight. These subscales can be used to calculate the overall eating disordered index, where values above 4 are treated in screening situations as indication of an eating disorder. The EDE-Q also measures frequency data on key behavioural features of eating disorders, such as the incidence of compensatory behaviours after meals. The questionnaire has been normed for young women (Mond, Hay, Rodgers, & Owen, 2006) and undergraduates (Luce, Crowther & Pole, 2008).

The Eating Attitudes Test (EAT, range 0-78; Garner, Olmstead & Polivy, 1982) assesses eating behaviours and body shape and size dissatisfaction. This is another commonly used screening questionnaire, with values over 20 considered to suggest the presence of an eating disorder (Garner et al., 1982). While EAT is particularly good at identifying individuals with the restrictive subtype of anorexia, its validity for the purging subtype of anorexia and bulimia has been brought in to question (Mintz & O'Halloran, 2000).

The 16-item Body Shape Questionnaire (BSQ, range 0-96; Evans & Dolan, 1993) assesses individuals' body size and shape concerns. It focuses particularly on preoccupations and attitudes towards body weight and shape. This commonly used screening questionnaire treats values over 66 as a sign of severe body dissatisfaction (Evans & Dolan, 1993)

Finally, in this thesis, participants' levels of depression and self-esteem were measured using the standardised questionnaires, the Beck Depression Inventory (BDI, range 0-63; Beck, Ward, Mendelson, Mock & Erbaugh, 1961) and the Rosenberg Self-Esteem Scale (RSE, range 0-30; Rosenberg, 1965), respectively.

Chapter 3

Experiments 1 and 2

The aim of these experiments was to test for normal perceptual biases that we should expect to be present in the size estimation of human bodies by healthy control participants.

3.1. Introduction

In the discussion of contraction bias in the Introduction (section 1.8), we quote Poulton (1989): “ ... *contraction biases can affect any kind of quantitative judgement or rating. In the absolute version of the stimulus contraction bias, magnitudes larger than the observer’s reference magnitude are underestimated. Magnitudes smaller than the observer’s reference magnitude are over-estimated*”. With respect to judgements about bodies, our understanding is that this would usually imply a single observer making judgements about others’ bodies. However, the context in which Cornelissen et al. (2013) raised the possibility of contraction bias was in relation to judgements of self. Therefore, one of the first experiments we felt was needed in this thesis was to check whether contraction bias can be confirmed when observers are making judgements about other people’s weights, as we should expect.

While contraction bias may go some way to explaining the over-estimation of very light weight bodies and the under-estimation of over-weight and obese bodies, there is another perceptual phenomenon described by Weber’s law, which means it should also get progressively more difficult to detect changes in body weight/size as the stimulus bodies become heavier. Weber’s law states that the just noticeable difference (JND) between two stimuli will be a constant proportion of their

magnitude, leading to a constant Weber fraction over the stimulus range (i.e. $\Delta I / I = K$, where I = stimulus magnitude and K = constant) (Gescheider, 1997). This means that it should be easier to notice, for example, a one BMI unit difference between two low BMI bodies than between two high BMI bodies. Over the full range of BMI, discriminating between higher BMI bodies should require progressively larger differences in BMI between stimuli. To our knowledge, there are no published studies to confirm whether the expectations from Weber's Law hold for body size estimations. Therefore, Experiment 2A and B test these basic predictions.

3.2. Experiment 1: Contraction Bias

Experiment 1 investigated whether a group of healthy control participants would show contraction bias when estimating the body weights of others. The computer program for this study was written by Andre Bester and was used at two different sites: Northumbria University and Newcastle University. Lucinda Gledhill who was at the time a PhD student, supervised by Dr. Martin Tovée, collected data using the program in Newcastle University, and Katri Cornelissen collected data in Northumbria University. The data collected at Northumbria University is reported as a part of this thesis, and the data collected at Newcastle University is reported as a part of Lucinda Gledhill's thesis (M. Tovée, personal communication, June 2nd, 2016).

3.3. Methods

3.3.1. Participants

We recruited 29 female participants (age $M = 30.9$, $SD = 9.3$) through opportunity sampling from staff and students at Newcastle and Northumbria

Universities in the UK. During recruitment we asked all potential participants whether they had a current diagnosis of an eating disorder or any history of such a disorder and excluded those individuals from this study.

3.3.2. Stimuli

One hundred and twenty digital photographs of female bodies were used as stimuli in this study. They were selected from the database of images reported in Tovée et al. (1999). To generate the images, consenting women were photographed standing in a set pose, front-facing, against a fixed dark background, at a standard distance from the camera, wearing tight grey leotards and leggings so that adiposity could easily be assessed by the observers. The images were stored as 24-bit colour pictures. The faces of the women in the images were blurred to protect anonymity. The women in the set of images for the current study varied in: weight from 28.2kg to 104.9 kg ($M = 59.14$, $SD = 13.44$); height from 1.47m to 1.83m ($M = 1.66$, $SD = 0.07$); BMI from 11.5 to 41.1 ($M = 2.56$, $SD = 4.87$).

3.3.3. Procedure

Before testing began, participants were informed that they were going to be shown a series of bodies varying in adiposity which they were required to estimate for weight using either kilograms or stones. They were free to use either scale depending on which one they were most comfortable with. Participants were then presented all 120 stimulus images, one at a time and in a randomized order, on a 19" flat panel LCD screen (1280w x 1024h pixel native resolution, 32-bit colour depth). Each image appeared on a plain black background beneath which was a linear scale

ranging from ~25kg-115kg (first mark 30kg, last mark 110 kg, 10kg increment) above the line and ~4st-18st (first mark 5st, last mark 17st, 1st increment) below the line. On each trial, with no time limitations, participants had to move a slider along the scale, and click with a mouse button when the participant judged that the slider had reached a weight corresponding to the weight of the woman in the image.

3.4. Results

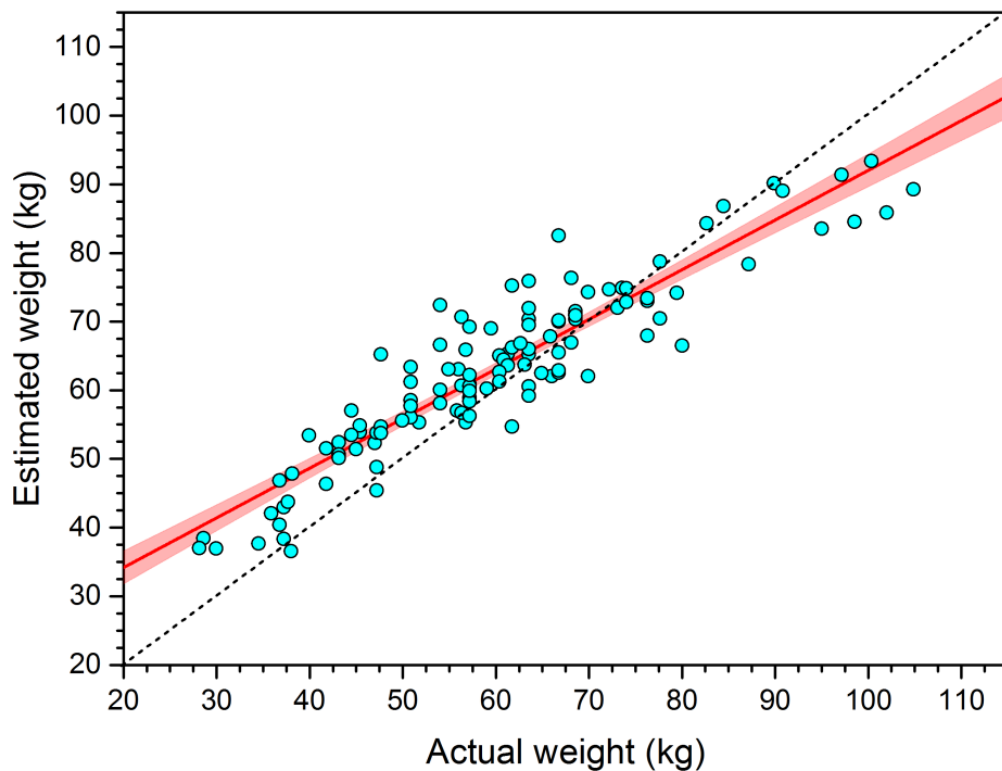


Figure 3.1. Scatterplot depicting the relationship between the actual weight of the women in the images (kg) and the mean of the participants' estimations of their weight (i.e. one data point represents one image). The red line represents the linear regression of estimated weight on actual weight and the pink shaded region its 95% confidence limits. The dotted black line represents the line of equality (i.e. slope unity, intercept zero).

In Figure 3.1 the dotted line represents veridical performance, if observers estimated body weight with perfect accuracy. However, Figure 3.1 shows clearly that there is a systematic departure from veridical performance. Images of women weighing ~70kg, i.e. the population average for Caucasian females in the UK (Health Survey for England, 2012), are estimated the most accurately. Thereafter, as the weight of women in the stimuli decreases, so participants systematically over-estimate their weight. Above the population average, observers systematically under-estimate body weight as the weight of the women in the stimuli increases.

We used PROC REG in SAS v9.3 to compute an ordinary least squares regression of mean estimated weight for each image as a function of actual weight. The assumptions for linear regression were met, and this model explained 85% of the variance in estimated weight. The overall model fit was statistically significant, $F(1,118) = 726.88$, $p < .0001$. (NB all p-values reported henceforth are for two-sided tests). The regression parameters, $\beta_0 = 19.60$, $t = 11.99$, $p < .0001$, CI [16.37 - 22.84] and $\beta_1 = 0.71$, $t = 26.96$, $p < .0001$, CI [0.66 - 0.76], showed a statistically significant, positive linear relationship between stimulus weight and estimated weight. However, the slope of this relationship was significantly less than 1, $F(1,118) = 124.18$, $p < .0001$. Because we had also measured the height of the women in the stimulus images, we could carry out an equivalent analysis of estimated BMI as a function of actual BMI. This model explained 82% of the variance in estimated BMI. The overall model fit was statistically significant, $F(1,118) = 552.51$, $p < .0001$. The regression parameters, $\beta_0 = 5.39$, $t = 7.01$, $p < .0001$, CI [3.87 - 6.91] and $\beta_1 = 0.79$, $t = 23.51$, $p < .0001$, CI [0.73 - 0.86], showed a statistically significant, positive linear relationship between stimulus weight and

estimated weight. The slope of this relationship was also significantly less than 1, $F(1,118) = 36.93$, $p < .0001$. These data therefore demonstrate convincing evidence for contraction bias when female observers judge the body weight of other women.

3.5. Experiment 2: Just Noticeable Difference (JND)

Experiment 2 investigated whether the just noticeable difference for body size estimation, made by healthy control participants, would be consistent with Weber's law. The data for this study was collected, analysed and is reported exclusively by Katri Cornelissen in this PhD thesis. However, for the journal article where this study is reported, both Experiments 1 and 2 from this chapter are included and Dr. Martin Tovée and his PhD student Lucinda Gledhill named as co-authors.

3.6. Methods

3.6.1. Participants

We recruited 28 female participants (mean age: 31.8; SD: 7.8) for this study from staff and students at Northumbria and Newcastle Universities in the UK. As in Experiment 1, we asked all potential participants whether they had a current diagnosis of an eating disorder or any history of such a disorder and excluded those individuals from this study.

3.6.2. Stimuli

Experiment 2 comprised two parts. In the first part, we wanted to identify the smallest change in BMI that observers could detect, i.e. the just noticeable difference (JND), at eight separate points along the BMI continuum from ~12 to ~45. The 8

points correspond to the boundaries between BMI categories (defined by the World Health Organization) as well as points within each category. As described in the Methods section, Chapter 2, we used CGI stimuli for this. However, we also wanted to make sure that our results were not confounded by the particular choice of 3D model, and the body shape morphs that are implemented in Daz 3D Studio. For this reason we created two stimulus sequences. For the first, we used the Victoria 5 model to which we applied the Genesis whole body shape morphs. For the second, we used the Victoria 6 model to whom we applied the Genesis 2 whole body morphs. We predicted that we would not find statistically significant differences related to model choice.

In the second part of Experiment 2 we identified the smallest change in BMI that observers could detect (i.e. the JND) at BMI values around 15, 18, 21 and 24, using the same database of images of real women that we used in Experiment 1. Clearly, by using natural images, there is a much wider variety of shape and height, and we wanted to be sure that effects from the CGI stimuli could be replicated in images of real women.

3.6.3. Procedure

For both parts of Experiment 2, in order to measure observers' JNDs, we used a 2-alternative forced choice (2AFC) discrimination paradigm, based on the method of constant stimuli. The images were presented on a 19" flat panel LCD screen (1280w x 1024h pixel native resolution, 32-bit colour depth). On every trial, participants were presented a pair of images, side by side, and were asked to respond by button press which of the pair (left or right) represented a thinner body. In the first part of Experiment 2, where we used the CGI stimuli, we presented observers 8

blocks of stimuli corresponding to the 8 points along the BMI continuum. Within each block, we presented pairs of images at each of 10 levels of BMI difference between the left and the right images. The set of differences in BMI between the image pairs was 0 to ± 2.5 BMI units in 0.25 BMI steps. The stimulus image pairs were therefore drawn from the 8 BMI ranges: 14.5-18.5; 16.5-20.5; 20-24; 23-27; 24.5-30.5; 27-33; 32-38; 37-43. Every image pairing, which represented a given BMI difference, was presented 20 times to each observer in order that we could calculate the probability that participants could detect that BMI difference.

In the second part of Experiment 2, we used the same database of images as was used in Experiment 1. We used a similar logic for the experimental procedure, but this time drawing images from only four BMI ranges; 15.0-17.5; 18-20.5; 21-23.5; 24-26.5. All four image sets covered a BMI range of 0 to 2.5 BMI units in 0.5 BMI steps. Clearly this represents a coarser grained procedure than was the case for the CGI images, but it was necessary because of the wide natural variation in shape and BMI in real images of women.

For both the CGI and real image experiments, we randomized the order in which stimuli within a given BMI block were presented to participants, as well as the order of presentation of the BMI ranges themselves. For each participant, we used probit analysis to fit psychometric functions and we defined the JND as the BMI difference between image pairs at which observers correctly identified the larger body 75% of the time. These values were compared across participants, as a function of BMI, to test for Weber's law behaviour.

3.7. Results

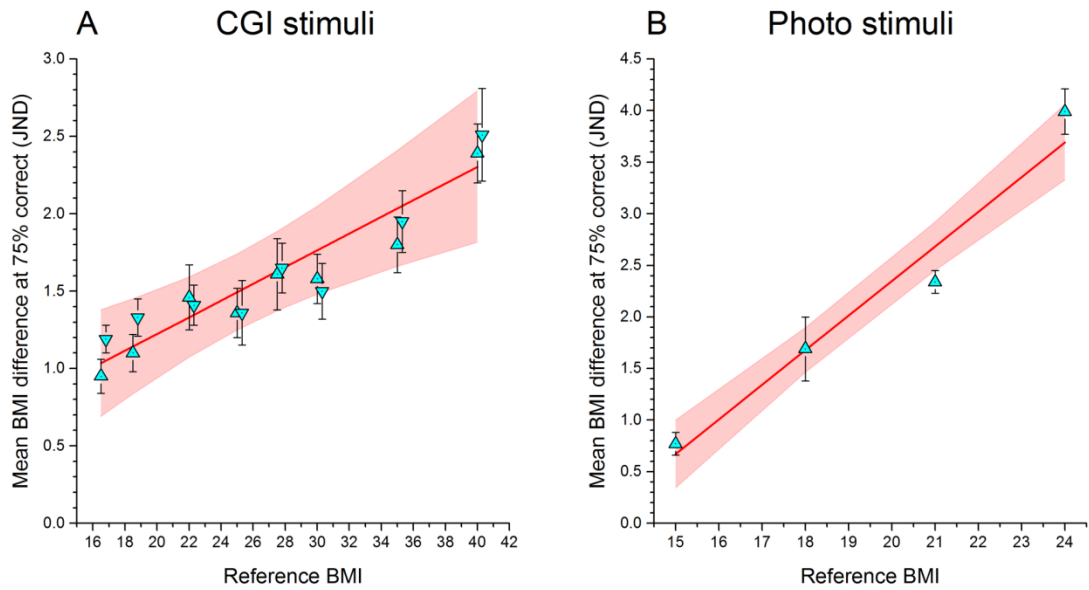


Figure 3.2: A) is a plot of mean JND as a function of the reference BMI value for each of the BMI ranges for the two CGI 3D models. The two models are indicated by the upward and downward pointing cyan triangles, respectively. Error bars represent 1 s.e. of the mean. The solid red line represents the main effect of BMI on JND derived from the mixed models (see text for details), and the pink shading its 95% confidence band. B) is an equivalent plot of mean JND as a function of the reference BMI value for each of the BMI ranges for the photos of real women. The Weber fractions in A were: 6.24% and 5.75% at BMIs 16.5 and 40, respectively; in B the Weber fractions were 4.53% and 15.33% at BMIs 15 and 24 respectively.

Figure 3.2A shows the mean JND at the 75% correct response rate plotted as a function of the centre BMI of the 8 BMI ranges for the two CGI images. We used PROC MIXED in SAS v9.3 to quantify the relationship between JND, reference BMI and model. The threshold data required transforming to ensure that they conformed to a normal distribution. We permitted individual slope and intercept variation for each subject by specifying an ‘unstructured’ variance-covariance

matrix. The mixed model explained 81% of the variance in mean JND. The Type III test of the fixed effect of BMI was statistically significant, $F(1,196) = 89.39$, $p < .0001$, CI [0.02 - 0.03]. However, neither the fixed effect of model nor the interaction between model and BMI were statistically significant: $F(1,71.2) = 3.12$, $p = .08$, CI [-0.03 - 0.40] and $F(1,196) = 1.98$, $p = .16$, CI [-0.01 - 0.003], respectively. For the real images in Experiment 3.2B, the equivalent mixed model explained 81% of the variance in mean JND. The Type III test of the fixed effect of BMI was statistically significant, $F(1,86.5) = 44.57$, $p < .0001$, CI [0.05 - 0.11].

3.8. Discussion

In Experiment 1, female participants estimated the weight of 120 women varying in their body mass. Their estimates clearly show contraction bias with bodies above 70kg being increasingly under-estimated and bodies below 70 kg being increasingly over-estimated. A value of 70 kg is the average body weight for adult women in the UK (HSE, 2012), and its adoption as a reference value against which to judge other female bodies would be consistent with people's visual diet shaping their reference body so that it reflects the population norm. As the height and weight of the women in the photographs is known, it is possible to calculate both the actual BMI of the women in the photographs and the BMI of their bodies based on the participants' estimation of their weight. These data show the same pattern of contraction bias, with a BMI of 27 being the most accurately judged, again consistent with a reference template based on the average BMI for adult women in the UK (HSE, 2012). Contraction bias can also be used to explain the accuracy of judgements of own body size over a wide range of BMI values from emaciated to

obese (Cornelissen et al., 2013) and can also explain the fact that previous studies have consistently shown that obese people under-estimate their size relative to normal weight people (Kuchler & Variyama, 2003; Kuskowska-Wolk & Rössner, 1989; Maximova, McGrath, Barnett, O'Loughlin, Paradis, et al., 2008; Robinson & Kirkham, 2013; Truesdale & Stevens, 2008; Wetmore & Mockdad, 2012).

In Experiment 2, participants had to judge which body was the heavier in a 2-alternative forced choice paradigm. Experiment 2 (part 1) demonstrated that Weber's law clearly applies to body judgements of the CGI models (i.e. a larger rise in BMI is required in higher BMI bodies to be detected than in lower BMI bodies), as we should expect. The CGI bodies accurately simulate the pattern of fat shown in real bodies (Crossley et al., 2013) and are judged in the same way as real bodies (Tovée, Edmonds & Vuong, 2012). The use of artificial bodies allows features such as height, body proportions, skin colour and texture to be held constant and the effect of increasing body fat to be directly modelled. By using 2 different body models and two different body fat simulations, the possibility that the results are an artefact of the CGI simulation of body fat increase was eliminated. Both parts of Experiment 2 showed that the ability to discriminate a difference in BMI became progressively worse as the BMI of the bodies being judged increased, as should be expected from Weber's law. However, only for the CGI stimuli did the Weber fraction (i.e. the JND in BMI units divided by the BMI of the bodies being judged) remain reasonably constant across the BMI range tested, and therefore only the results from the CGI stimuli conformed fully to Weber's law. This is probably because adiposity was the only salient change for these stimuli as BMI increased. While it is true that the JND did increase as a linear function of BMI for the photo stimuli, consistent with Weber's law, nevertheless the Weber fraction accelerated with BMI in Experiment

2b, from 4.53% to 15.33% over the BMI range of those stimuli – it did not remain constant. In motor control, it is the absolute noise in the system that determines the precision with which a task can be achieved – an increase in absolute noise causing a decrease in motor precision (e.g., van Beers, Baraduc & Wolpert, 2002). In principle, this would be reflected by an increase in the Weber fraction for, for example, haptic perceptual tasks. Such tasks require participants to make judgements that require a force-movement-interaction between their hand(s) and the object being manipulated. Typically, the participant applies forces to the object, which generally leads to reaction forces that act on the perceiver's hand(s). Another example in which non-scalar changes in Weber fractions have been convincingly demonstrated empirically is in interval timing for periods between 1 and 2 seconds (Grondin, 2012). In this case, Grondin proposed that the time span from 1 to 2 seconds may bracket a critical point in time (~1.5s) when a different temporal mechanism comes in to play, transitioning from a more precise internal clock mechanism to a less precise temporal chunking strategy. We suggest that for photographs of real women, there may be increases in the absolute noise of the visual stimuli (i.e. individual shape variation across different images of women at different BMIs) for at least 2, if not more, sub-ranges of BMI. This may therefore lead to successive reductions in the perceptual precision with which body size estimates can be made, reflected in increasing Weber fractions. To illustrate why, one possibility is that for women with very low BMI, most shape variation across individuals will depend primarily on underlying skeletal structure alone. However, for much larger women where the skeletal outlines are no longer visible, it is possible that the variation in body shape and skin folds that occur with increasing adiposity may be, proportionately, much greater than the linear increase in BMI would otherwise suggest.

Chapter 4

Experiment 3

In this study we explored the inter-relationships between body size estimation, an individual's BMI and their psychological concerns about body shape, weight and eating, in 42 women with a history of anorexia nervosa and 100 healthy control participants.

4.1. Introduction

At the end of section 1.11 in the main Introduction to this thesis, we argued that a fuller understanding of body image distortion in anorexia nervosa will likely need to take account of *observer* BMI, because of perceptual effects like contraction bias, and, simultaneously, the parallel contributions from the psychometric dimension. Experiments 1 and 2 in the preceding chapter confirm the need to include observer BMI in any adequate model.

According to the “single channel” model presented in the Introduction (see Figure 1.5, page 57), contraction bias would be a sufficient explanation for the perceptual component of BID in women with AN. The re-analysis of the data in Tovée et al. (2003), reported in Cornelissen et al. (2013), was consistent with this idea because the same slope (<1) and intercept values captured the regression of estimated BMI on BMI for *both* the AN and the control participants, as is illustrated in Figure 4.1A overleaf.

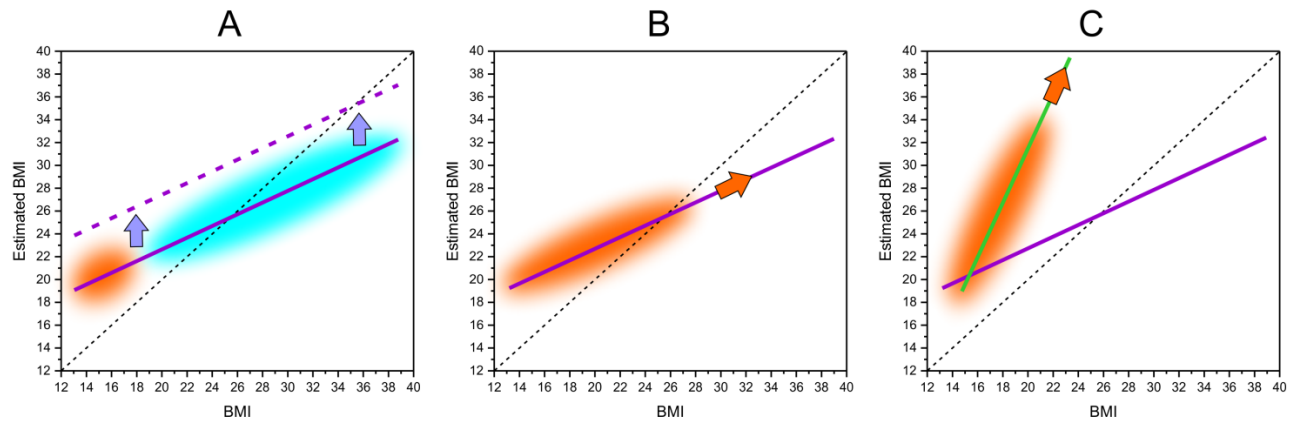


Figure 4.1 A) Schematic representation of the results from Cornelissen et al. (2013) in which women with AN and controls used an interactive software program to estimate body size. The line of equality (i.e. perfect accuracy) is shown by the dotted black line. The response distribution of control participants is shown in blue and that of AN participants in orange. The solid purple line represents the regression of estimated BMI on BMI and has the same slope and intercept for women with AN and controls. The dashed purple line represents the increase in intercept for this regression line as psychological concern about body shape and weight increase. B) The pattern of body size estimation predicted by the contraction bias model in women with AN, or recovering from AN (i.e. in an eating disordered group with a wider BMI range). C) If increasing psychological concerns drive body size over estimation, rather than contraction bias, as in B), then the regression of estimated BMI on BMI should follow the green trajectory in women with AN, as their BMI increases.

However, an important limitation to this result is that the BMI of the women with AN in Tovée et al. (2003) fell within a relatively narrow range of 11.5 to 18.4 (6.9 BMI units). In contrast, most of the variation in BMI belonged to the control

participants, ranging between 14.7 and 36.8 (22.1 BMI units). If the “single channel” model is a sufficient explanation for body size over-estimation in AN, then, with a wider BMI range (including recovering patients to expand the range), the responses of these women should follow the same pattern as healthy controls. This is illustrated in Figure 4.1B, where the orange arrow predicts that the regression of estimated BMI on actual BMI in these women should track up along the same regression line as in Figure 4.1A, when BMI increases. In other words, if the “single channel” model is correct, as BMI increases in women with AN, so body size over-estimation should *decrease*.

Alternatively, psychological / attitudinal factors may constitute the primary driving force behind body size over-estimation in women with AN. This view is entirely consistent with reports that an individual’s body size (as indexed by BMI) is strongly correlated with body dissatisfaction (Gardner, Brown, & Boice, 2012; Stice & Shaw, 2002; Striegel-Moore, Franko, Thompson, Barton, Schreiber, 2004). If so, we should predict a different outcome. For example, women with AN who have a very low BMI might be expected to have relatively low body size concerns, but during the recovery process as their weight increases, their body size concerns would rise in parallel. Therefore, as their weight increases, we should predict there is a rapid *rise* in the degree of body size over-estimation reflecting their accelerating concerns about body shape and weight. This is illustrated in Figure 4.1C, where the orange arrow shows how the regression of estimated BMI on BMI for women with AN should follow the trajectory of the solid green line.

To distinguish between these two hypotheses, in Experiment 3, we recruited a group of women all of whom had had a diagnosis of AN, but who were in various stages of recovery / treatment at the time of testing, and who demonstrated

significant variation in their BMI and psychological concerns. Because these individuals do not all conform to the strict criteria for AN in DSM 5, they should be more correctly referred to as suffering from anorexia spectrum disorders (ANSO) – the term to be used henceforth in this thesis. However, from an empirical point of view, we need to take advantage of individual differences in this way, in order to determine whether the accuracy of body size estimation increases or decreases as the BMI of ANSO participants varies and therefore determine the relative importance of psychological factors and perceptual bias as illustrated in Figure 4.1B and C.

4.2. Methods

The experimental procedures and methods for participant recruitment for this study were approved by: the local ethics committee at Northumbria University; the Beating Eating Disorders Organisation (BEAT) and the Northern Initiative on Women and Eating (NIWE) Organisation.

4.2.1. Participants

Forty two female participants who had had a formal diagnosis of anorexia nervosa ($n = 34$) or bulimia nervosa, followed by a diagnosis of anorexia nervosa ($n = 8$), according to DSM-IV-R or DSM-5, consented to take part in this study. In addition, we recruited 100 control participants from staff and students in and around Newcastle and Northumbria Universities, none of whom had a history of eating disorders. The samples of ANSO and control participants were deliberately permitted to have as wide a variation of body mass index (BMI) and attitudes to eating and body shape as possible. Participants were recruited using multiple channels, including using posters in Northumbria University buildings, website

advertising through eating disorder support organisations, advertising at the slimming groups, and through personal contacts.

4.2.2. Anthropometric measurements

We measured each participant's weight and height with a set of calibrated scales and a stadiometer, respectively, and used these values to calculate body mass index (BMI). We measured each participant's waist, hip, under-bust and bust circumference with measuring tapes. In addition, we used Harpenden Callipers to measure four skinfold thicknesses (tricep, bicep, subscapular, iliac crest). These were used to calculate each participant's percentage of body fat from a standard formula (Durnin & Womersley, 1974).

4.2.3. Psychometric measurements and procedure

Participants gave responses to all the questionnaires as outlined in the Methods section 2.3. These were: (i) the Eating Disorders Examination Questionnaire (EDE-Q); (ii) the Eating Attitudes Test (EAT); (iii) the 16-item Body Shape Questionnaire (BSQ); (iv) the Beck Depression Inventory (BDI); (v) the Rosenberg Self-Esteem Scale (RSE).

4.2.4. Psychophysical measurements and procedure

We used the method of constant stimuli, configured as a 2AFC task as outlined in the Methods section 2.2.1, in order to estimate participant's PSE and DL.

4.3. Results

4.3.1 Reliability measures

The questionnaire responses as well as scores from the psychophysical experiment were analysed for reliability. Cronbach's alpha was used to estimate the internal consistency of the questionnaire scores. Excellent internal consistency was found for all body shape and eating attitude questionnaires: .96 for EDE-Q, .92 for EAT and .96 for BSQ. Cronbach's alpha for the questionnaire measuring depression (BDI) was .93, and .89 for the questionnaire measuring self-esteem (RSE).

We estimated the reliability of PSE scores from the 2AFC task in two ways. Firstly, the PSE scores for the last block from the psychophysical experiment were divided into two halves. A PSE score was estimated separately for each half. The correlation between the first and second half PSE scores was then computed. We found a statistically significant Pearson correlation ($r=.92$; $p<.0001$) between the first and second half scores across the whole sample, suggesting good reliability. Secondly, 17 participants were asked to return to the lab a minimum of four weeks after initial testing and to complete both the psychophysics task and to have their BMI measured. The participants' BMIs were not significantly different in a dependent t-test comparison of the means (BMI 1 $M=22.90$, $SD=4.88$; BMI 2 $M=22.72$, $SD=4.82$; $t_{16}=1.03$, $p=.319$), neither were their PSE scores (PSE 1 $M=22.20$, $SD=3.62$; PSE 2 $M=21.72$, $SD=4.79$; $t_{16}=1.00$, $p=.333$). The Pearson correlation between the two PSE estimates was $r=.93$ ($p<.0001$).

4.3.2. ANSD participant characteristics

First, we wanted to analyze the heterogeneity of the ANSD participants. The choice of including participants at different stages of recovery with different body sizes was deliberate, and was needed in order to have sufficient within group variability to distinguish between the "single channel" and "dual channel" models.

Table 4.1 shows the details of the ANSD participants divided into 3 subgroups: ANSD1: women (n=12) who were currently in outpatient treatment with a diagnosis of anorexia nervosa; ANSD 2: women (n=20) with a diagnosis of anorexia nervosa, but who were currently no longer in treatment; ANSD 3: women with a diagnosis of either atypical AN (n=3) or Bulimia that switched to AN (n=8), 4 of whom were being treated at the time of testing. The right hand columns of Table 4.1 show the output of pairwise comparisons of the three subgroup means, adjusted for multiple comparisons, using the permutation method in PROC MULTTEST (SAS v9.3).

Table 4.1: Characteristics of the three ANSD subgroups in Experiment 3.

	ANSD1 (n=12)		ANSD2 (n=20)		ANSD3 (n=10)		ANSD 1v2	ANSD 1v3	ANSD 2v3
	<i>M</i>	<i>SD</i>	<i>M</i>	<i>SD</i>	<i>M</i>	<i>SD</i>	<i>p</i>	<i>p</i>	<i>p</i>
Participant characteristics									
Age (years)	22.42	3.65	25.80	7.62	22.36	3.44	.78	.99	.81
BMI (weight/height ²)	18.40	2.35	21.03	2.95	22.62	1.59	.12	<.01	.63
Age at onset (years)	17.42	3.92	15.55	2.01	16.45	1.97	.54	.99	.92
Treatment ended (years)	0.00	0.00	3.45	4.11	1.95	2.90	-	.23	.96
Duration of illness (years)	5.04	4.70	6.75	6.79	4.91	3.05	.99	.99	.99
Depression and self esteem									
RSE score	8.33	2.35	16.58	5.03	15.70	5.81	<.01	.02	.99
BDI score	31.91	9.49	15.74	8.10	15.00	10.46	<.01	.01	.99
Eating & body shape concern									
EAT score	39.25	15.70	21.10	13.27	28.80	15.51	.01	.72	.80
BSQ score	71.00	17.69	55.50	17.00	62.20	18.47	.16	.95	.98
EDE-Q score	4.01	1.34	2.75	1.18	3.05	1.65	.09	.76	.99
EDE-Q wc score	4.08	1.58	3.05	1.34	3.70	1.50	.40	.99	.92
EDE-Q bs score	4.70	1.35	3.77	1.41	3.81	1.58	.50	.81	.99
Psychophysical performance									
PSE (weight/height ²)	20.57	4.93	21.95	5.26	23.66	4.15	.99	.66	.98
OE i.e., PSE – BMI	2.17	3.25	1.10	2.83	1.04	3.03	.99	.77	.93
DL (weight/height ²)	0.53	0.28	0.65	0.46	0.98	1.05	.97	.99	.99

Note. ANSD1: AN participants being treated at the time of testing. ANSD 2: AN participants no longer being treated; ANSD 3: atypical AN or Bulimia that switched to AN. BDI = Beck Depression Inventory. RSE = Rosenberg Self-Esteem Scale. EAT = Eating Attitudes Test. BSQ = Body Shape Questionnaire. EDE-Q = Eating Disorder Examination Questionnaire global score. EDE-Q wc = Eating Disorder Examination Questionnaire weight concern subscale. EDE-Q bs = Eating Disorder Examination Questionnaire body shape concern subscale. PSE = Point of Subjective Equality. OE = Over Estimation. DL = Difference Limen.

The analysis of the subgroups shown in Table 4.1 reveals tendencies consistent with previous research (e.g., Tovée et al., 2003; Tovée, Emery, Cohen-Tovée, 2000). Individuals who are currently in treatment do have lower BMIs

consistent with their ongoing clinical need. The individuals who were currently in treatment also had lower self-esteem, indicated by their RSE scores, higher tendency towards depression, indicated by BDI scores, and the higher scores in all measures investigating body shape and size concerns. This was expected, as in the clinical setting these were the individuals who were considered most in need of treatment and who are at an earlier stage of recovery. Despite these differences, no significant differences were found in the psychophysical measures PSE and DL, nor in the magnitude of body size overestimation (i.e. PSE - BMI).

A key decision needed to be made whether these subgroups should be retained for the comparisons with control participants. Therefore, we tested the effect of ANSD subgroup on the relationships between PSE and BMI as well as between DL and BMI using PROC MIXED (SAS v9.3). The DL scores were log transformed prior to the analysis because their distribution was non-normal (Shapiro-Wilk's $W=.62, p < .001$). For PSE, the mixed linear model explained 71% of its variance. The fixed effect of BMI was significant at the specified .05 level, $F(1,37) = 67.36, p < .001$, but the tests for ANSD group, $F(2,37) = 0.55, p = .580$, and the interaction between BMI and ANSD group, $F(2,37) = 0.54, p = .587$, were not. For DL, the mixed linear model explained 15.8% of its variance. The fixed effect of BMI was significant at the specified .05 level, $F(1,37) = 7.11, p = .011$, but the tests for ANSD group, $F(2,37) = 1.09, p = .347$, and the interaction between BMI and ANSD group, $F(2,37) = 1.07, p = .354$, were not. In the absence of any influence of sub-group, and to ensure greater statistical power for the final analyses, we treated the ANSD participants as a single, uniform group henceforth.

Table 4.2, overleaf, shows the characteristics for the combined ANSD participants and the healthy controls. Consistent with prior research, ANSD

participants had significantly lower BMI and percentage body fat in both measures of percentage body fat. Furthermore, ANSD participants on the whole had significantly higher scores in all questionnaires investigating body size and shape concerns (EDE-Q, EAT, BSQ), a greater tendency towards depression (BDI), and lower self-esteem (RSE).

Table 4.2: Means and standard deviations for the participant characteristics, separated according to whether they belong to the ANSD or the healthy control group in Experiment 3.

	ANSD (<i>n</i> =42)		Control (<i>n</i> =100)		<i>p</i>
	<i>M</i>	<i>SD</i>	<i>M</i>	<i>SD</i>	
Participant characteristics					
Age (years)	23.80	6.05	24.02	8.91	.99
BMI (weight/height ²)	20.89	2.84	24.01	5.03	<.01
Percentage body fat	25.56	6.41	31.63	5.04	<.01
Depression and self esteem					
BDI	20.00	11.37	9.09	6.08	<.01
RSE	14.00	5.96	18.56	4.79	<.01
Eating and body shape concern					
EAT	27.38	15.55	11.07	9.08	<.01
BSQ	61.10	18.63	50.08	17.33	<.01
EDE-Q	3.12	1.42	2.01	1.21	<.01
EDE-Q wc	3.50	1.48	2.21	1.44	<.01
EDE-Q bs	4.04	1.46	2.97	1.50	<.01
Psychophysical performance					
PSE (weight/height ²)	22.21	4.95	23.80	5.13	.36
OE (i.e., PSE – BMI)	1.41	3.04	-0.14	2.85	.04
DL (weight/height ²)	0.72	0.66	0.99	0.81	.32

Note. BDI = Beck Depression Inventory. RSE = Rosenberg Self-Esteem Scale. EAT = Eating Attitudes Test. BSQ = Body Shape Questionnaire. EDE-Q = Eating Disorder Examination Questionnaire global score. EDE-Q wc = Eating Disorder Examination Questionnaire weight concern subscale. EDE-Q bs = Eating Disorder Examination Questionnaire body shape concern subscale. PSE = Point of Subjective Equality. OE = Over Estimation. DL = Difference Limen.

In a pairwise comparison, ANSD participants showed significantly greater over-estimation of body size (OE), which is in line with most previous research (Cash & Deagle, 1997; Gardner & Bokenkamp, 1996). Furthermore, there was no significant difference in the sensitivity to body size differences, measured by DL, which was expected on the basis of the earlier literature and effect size measures (Cash & Deagle, 1997).

4.3.3. Comparisons between ANSD and control participants

To model the body size estimation data, PROC MIXED (SAS v9.3) was used to build separate mixed linear models with estimated BMI (i.e., PSE) and task sensitivity (i.e. DL) as the outcome variables, and actual BMI and GROUP (i.e., ANSD versus healthy controls) as predictor variables. To estimate the influence of attitudinal component of body size estimation we wanted to include the psychometric questionnaire scores. However, as statistically significant correlations were found between these psychometric responses, which would lead to variance inflation if all the questionnaires were included in the models, an iterated principal factor analysis with rotation was carried out to identify latent psychometric variable(s). (The Pearson correlations between RSE and BDI, RSE and BSQ, RSE and EAT, RSE and EDE-Q, BDI and BSQ, BDI and EAT, BDI and EDE-Q, BSQ and EAT, BSQ and EDE-Q, and EAT and EDE-Q were: -.75, -.61, -.62, -.57, .60, .66, .56, .72, .84 and .79 respectively; all statistically significant at $p < .001$). A single latent variable (PSYCH) was identified which represents concerns over eating, body size and shape, and low self-esteem and depression, i.e., the attitudinal component of body image distortion. The Kaiser-Meyer-Olkin (*KMO*) measure of sampling adequacy (which indicates the degree of diffusion in the pattern of correlations) was .81 suggesting an acceptable sample. PSYCH had an Eigen value greater than

Kaiser's criterion of 1 (i.e., 3.39) which explained 98% of the variance. The scree plot showed an inflexion, i.e., Cattell's criterion which also justified retaining just the one factor PSYCH. The residuals were all small, and the overall root mean square off-diagonal residual was 0.081, indicating that the factor structure explained most of the correlations. The factor loadings for RSE, BDI, BSQ, EAT and EDE-Q were: -.76, .76, .86, .86, .86 respectively. The influence of AGE was also controlled in the following statistical models. Moreover, we note that the Pearson correlations between BMI and PSYCH for the ANSD and control participants were $r = -0.09$, $p = .584$ and $r = 0.19$, $p = .068$ respectively.

4.3.3.1. PSE

The mixed linear model for PSE explained 71% of its variance. Both ANSD and control participants showed a positive, linear relationship between actual BMI and PSE, $F(1,137) = 207.72$, $p < .001$, 95% *CI* [0.68, 0.90]. Critically, however, the pattern of estimation by women with ANSD is statistically different from that of the controls because we found a significant main effect of GROUP, $F(1,137) = 14.90$, $p < .001$, 95% *CI* [-19.08, -6.16] as well as an interaction between GROUP and BMI, $F(1,137) = 15.33$, $p < .001$, 95% *CI* [.29, .89].

The PSE difference between the groups is illustrated in Figure 4.2 overleaf. As can be seen from Figure 4.2A, low BMI control participants over-estimate their body size, whereas individuals with high BMI under-estimate their body size, and those within the healthy BMI are the most accurate on a whole, consistent with contraction bias (Poulton, 1989). The regression slope for this relationship was significantly less than 1, $\beta = 0.82$, $F(1,97) = 9.96$, $p < .005$. On the other hand, low BMI ANSD participants were very accurate in their perception, and high BMI individuals overestimated their body size, and this overestimation systematically

increased with increased BMI of the participant. The regression slope for PSE on BMI in ANSD participants was significantly greater than 1, $\beta = 1.39$, $F(1,39) = 5.86$, $p < .05$.

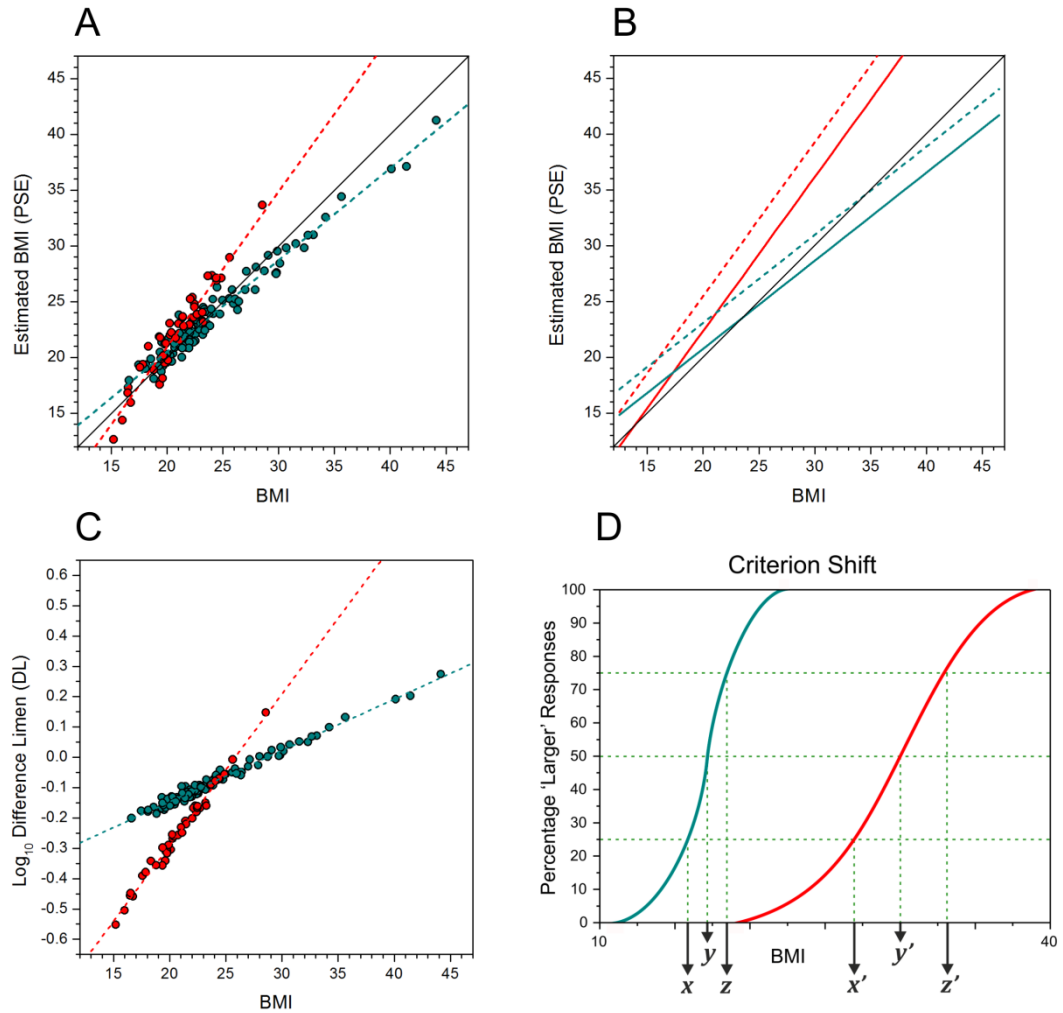


Figure 4.2: (A) Shows the relationship between participants' BMI (x-axis) and their subjective estimate of body size (PSE) with the effects of PSYCH statistically controlled. (B) Shows the relationship between participants' BMI (x-axis) and fitted values of estimated body size (PSE) computed from the mixed model at ± 1 SD of the mean PSYCH value for each group. (C) The relationship between participants' BMI (x-axis) and their sensitivity at estimating body size (Log₁₀ DL) with the effects of PSYCH statistically controlled. (D) A sketch plot to illustrate the relationship

between the psychometric function for body size estimation and the calculation of PSE (i.e., the value of BMI at y and y' when percentage 'larger' responses = 50%) and DL (i.e., the difference in BMI between z & x and z' & x'). These points correspond to values of BMI when percentage 'larger' responses equal 75% and 25%, respectively). Two situations are illustrated: accurate and sensitive performance (blue) versus over-estimating and insensitive performance (red).

The effect of the attitudinal component (PSYCH) on the body size estimation is illustrated in Figure 4.2B. As can be seen, the effect of PSYCH on PSE was statistically significant (Type III test of the fixed effect: $F(1,137) = 17.23, p < .001$, 95% CI [0.59, 1.66]), but also similar in both participant groups. This means that any participants with greater PSYCH score, irrespective of the participant group, were more likely to overestimate their own body size.

To explore the extent to which these results may have depended on ANSD participants with the highest BMIs, the model was refitted by reducing the participant number from the ANSD group by one at a time. Statistically significant results remained even though the sample size of ANSD participants was reduced from 42 to 28 which is 67% of the original sample. The model was also refitted using a reduced sample by removing all participants who had a second diagnosis of bulimia nervosa ($n = 8$). For this reduced model, the Type III tests of the fixed effects of BMI, $F(1,129) = 175.33, p < .001$, 95% CI [0.68, 0.90], PSYCH, $F(1,129) = 14.40, p < .001$, 95% CI [0.50, 1.60], GROUP, $F(1,129) = 10.85, p = .001$, 95% CI [-18.03, -4.50], and the interaction between BMI and GROUP, $F(1,129) = 11.03, p = .001$, 95% CI [0.22, 0.85], were all significant at the specified .05 level, suggesting

that these data are robust and not obviously sensitive to variation in the underlying clinical diagnosis.

4.3.3.2. DL

The mixed linear model for $\log_{10}DL$ explained 22.1% of its variance. Both ANSD and control participants showed a positive, linear relationship between actual BMI and $\log_{10}DL$, $F(1,137) = 21.98, p < .001, 95\% CI [0.0068, 0.025]$. However, the significant main effect of GROUP, $F(1,137) = 7.05, p = .008, 95\% CI [-1.32, -0.19]$ as well as the interaction between GROUP and BMI, $F(1,137) = 5.00, p = .027, 95\% CI [0.0034, 0.056]$ show that the pattern of estimation by women with ANSD is statistically different from that of the controls. As can be seen from Figure 4.2C control participants showed a reduced sensitivity (indicated by an increased DL) with increased BMI, consistent with Weber's law. ANSD participants showed the same pattern, but with greater extremes. The individuals with the smallest BMI showed very low DL values, indicating high sensitivity. However, as the ANSD participants' BMI increased, their DL values dramatically increased. The attitudinal component, PSYCH, was not related to $\log_{10}DL$ ($F(1,137) = 0.75, p = .389, 95\% CI [-0.026, 0.067]$).

4.4. Discussion

In Experiment 3 we explored the inter-relationships between participants' estimated BMI (i.e. PSE), actual BMI and the psychological concerns of women who had a diagnosis of anorexia nervosa and we compared them to the responses of healthy controls. To uphold the contraction bias model (Figure 4.1B), we should have found that increasing BMI in women with ANSD should lead to *reducing* body

size over-estimation, because their judgements should follow the same trajectory as has been shown for healthy controls. In contrast, to uphold the attitudinal / psychological concerns model (Figure 4.1C), we should have found that increasing BMI in women with ANSD should lead to *increasing* body size over-estimation.

In healthy control participants, we found that the accuracy of body size estimation is linearly predicted by participants' BMI, but with a slope less than unity (see Figure 4.2A): low BMI controls over-estimate body size, mid-range BMI controls' estimates are the most accurate and high BMI controls under-estimate. This result is consistent with contraction bias (Poulton, 1989), which is a normal feature of magnitude estimation. Moreover, the contraction bias explanation is consistent with results from other studies of patients with anorexia nervosa (Tovée et al., 2003) as well as studies of people suffering from obesity who under-estimate their size relative to normal BMI people (Kuchler & Variyama, 2003; Kuskowska-Wolk et al., 1989; Maximova et al., 2008; Robinson & Kirkham, 2013; Truesdale & Stevens, 2008; Wetmore & Mockdad, 2012).

For control participants, while the gradient of the regression of PSE on BMI was less than 1, consistent with contraction bias, the intercept for this relationship was also influenced by body shape and eating concerns, because the intercept depended on the PSYCH latent variable, illustrated in Figure 4.2B. Therefore, the over- or under-estimation of body size made by a non eating-disordered observer is a function not only of their actual BMI but also their psychological state.

We found that the difference limen (DL) for the control participants increased as the participants' own BMIs rose. This means that control participants' sensitivity to changing body size declines as their own BMI rises. Given the results

from Experiments 2A and B in this thesis, the result was also to be expected, and is entirely consistent with Weber's law. Further evidence in support of this explanation is the fact that the Weber fractions at, e.g., BMI 20 and 40 are 3.6% and 3.8% respectively, which fall within 5% of each other.

In Experiment 3, the performance of women with ANSD was qualitatively different from that of healthy controls. For women with ANSD, individuals with the lowest BMI were the most accurate in estimating body size. They were also more sensitive at detecting body size difference in low BMI bodies (i.e., a low DL) than healthy controls. However, as BMI increased in women with ANSD, they increasingly over-estimated their body size (as indexed by PSE) as a function of their BMI. At the same time, this was associated with a rapid increase in DL scores, consistent with a marked decline in sensitivity to changes in body size. The gradient of this relationship for DL in women with ANSD was substantially steeper than would be predicted by Weber's law because the Weber fraction did not remain approximately constant. Together, this pattern of results is markedly different from that in healthy controls, is the opposite of the pattern of responses predicted by contraction bias, and is more consistent with the prediction in Figure 4.1C.

The illustration in Figure 4.1C supposes that women with ANSD who have low BMI would also have relatively low body size concerns. As these individuals' BMI increases, so their psychological concerns should increase leading to ever increasing over-estimation of body size. We found that the effect of psychological concerns on the regression of estimated BMI on personal BMI was the same for both groups (see the difference in intercept between continuous and dotted regression lines in Figure 4.2B). Thus, increasing psychological concerns predicted higher body size estimations across the entire sample, elevating the intercepts, but not the slopes

of the regressions by the same amount for both groups. This means that differences in psychological concerns per se cannot be directly responsible for the perceptual differences in the two groups that we observe, i.e., the different slopes for PSE and DL in Figures 4.2 A and C. If this were true, we would have found statistical evidence for three way interactions between BMI, Group and PSYCH, and we did not. Instead, we suggest that the key to explaining our findings may lie in the different ways that women with ANSD *interpret* changes in body size and psychological concern, compared to controls. In the following account, we put the marked reduction in their task sensitivity centre stage, to act as the driver for body size over-estimation.

Reducing sensitivity in the 2AFC task (indexed by increasing DL) means that as the BMI of participants with ANSD increases, they become less able to distinguish small differences between the BMI of the stimuli and the body size they believe they have. Let us imagine two women who have anorexia, one with low BMI and the other high BMI, and we wish them to have the same confidence in their responses during the 2AFC. Because of the difference in sensitivity between these two individuals, the woman who has a higher personal BMI should only response ‘larger than me’ to stimuli that are, proportionately, considerably larger than they are, as compared to the participant who has a lower personal BMI. Psychophysically, this can come about through a so-called criterion shift: i.e., not only does the slope of the psychometric function become shallower for the participant with a higher personal BMI (i.e., reduced sensitivity), but the curve itself is also shifted to the right (see Figure 4.2D). Therefore, one way to explain the results is that because of their reduced sensitivity to changes in body size, the higher BMI ANSD participants shift their response criterion in order to remain confident that their responses are correct,

which in turn causes an increase in PSE and consequent over-estimation of body size. That women who have ANSD should insist on wanting to feel confident about the decisions they make in the 2AFC task is consistent with their documented aversion to making errors in judgements (Kaye et al., 2009; Wagner, Aizenstein, Venkatraman, Fudge, May, 2007). As a post-hoc analysis, we computed the correlation between psychological concerns in women with ANSD and both PSE and DL: $r = .30, p = .05$ and $r = .17, p = .29$ respectively. We then partialled out any potential influence of DL, and found that the correlation between psychological concerns and body size estimation was rendered non-significant ($r = .25, p = .11$). This therefore suggests that sensitivity to change in body size plays a critical role in modulating the relationship between psychological concern and body size estimation in women with anorexia.

The question that remains unanswered is: what mechanism might cause the dramatic differences in task sensitivity demonstrated by women with ANSD? The current experiment cannot address this question directly. However, one possibility is that women with a history of anorexia may develop a particular expertise effect at discriminating between very low BMI body shapes. A number of studies have shown that women with AN spend a great deal of time looking at low BMI bodies including their own, but also online as part of their obsession with the thin ideal (Norris, Boydell, Pinhas, & Katzman, 2006; Ransom, La Guardia, Woody, & Boyd, 2010). Therefore, repeated viewing and evaluation of low BMI bodies might lead to heightened expertise which would in turn be reflected in high sensitivity levels in the 2AFC task. It is noteworthy that the ANSD participants' sensitivity to size change in higher BMI bodies showed a rapid decline, far faster than is seen in the control participants. Since body shape changes in a non-linear fashion with increasing BMI

(Wells, Treleaven, & Cole, 2007), so the pattern of shape change with weight increase is different in low BMI bodies as compared to heavier BMI bodies. Therefore, the expertise developed by women with ANSD might be specific to low BMI bodies and may not generalise to discriminating between higher BMI bodies.

Finally, we note that Gardner and Moncrieff (1988) used Signal Detection Theory to analyse their data from a 2AFC task in which participants had to judge whether images of themselves were normal or distorted. Gardner and Moncrieff (1988) found that the women with AN showed a more lax response criterion (β) than their controls, but no difference in task sensitivity (indexed by d-prime). They proposed that this might cause women with AN to report an image of themselves as distorted more frequently than controls. Unfortunately, Gardner and Moncrieff (1988) do not report the BMIs of their participants, only their weights. However, assuming an average height of 1.6m, then the average BMI of the women with AN and controls in their study would have been ~ 17.5 and ~ 22 respectively. In the current study, we see striking differences in task sensitivity (indexed by DL) and PSE comparing women with ANSD and controls at these BMI levels. We therefore suggest three reasons for the differences between studies. The first is that both classical psychophysics measures, PSE and DL, can be influenced by participants' expectance or bias (Gescheider, 1997), so that there does not exist a direct one to one mapping between them and the signal detection theory measures, d-prime and β . Secondly, Gardner and Moncrieff (1988) used the video distortion technique (VDT) which, as illustrated in Figure 1.4 (page 47), generates systematic body shape differences, compared to our CGI method. Therefore, this might be sufficient to induce different patterns of response in women with ANSD and controls. Thirdly, it is possible that the use of a distortion question (i.e., normal versus distorted) may

have invoked a different response pattern from the women with AN than the thinner or fatter question we used and which is potentially a more psychologically and emotionally charged judgement for them to make. Clearly, further research is needed to clarify the nature and implications of these methodological differences.

In conclusion, our results suggest that body size over-estimation is not only much more variable than might have been appreciated previously, given the strong dependence on individual BMI, but it also shows striking differences between women with ANSD and controls. The accuracy of control participants can be explained by the combined effects of two perceptual functions, contraction bias and Weber's law, with a modulating effect of psychological concerns about body size. By comparison, as their BMI increases, the accuracy of estimation by women with ANSD seems to be predicted primarily by a rapid criterion shift which is to compensate for a rapid decline in sensitivity to body size change. However, we should sound a final note of caution. We used a correlational design in this study. Therefore, we cannot draw any causal inferences from the associations that we have found between perceptual and psychometric measures and participants' BMI. To do this, we would need data from prospective longitudinal studies in which for example, we could track BMI changes in AN patients and use this information to predict body size over-estimation and psychometric changes on time.

Chapter 5

Experiment 4

In this study we carry out a replication of Experiment 3 using avatars of individual participants created from 3D body scans.

5.1. Introduction

This chapter reports Experiment 4 which extended the body size estimation task by generating stimuli which are tailored to individual participants, being derived from 3D body scans of each individual, and we also investigated the ideal body size that participants would like to have. Thus far the psychophysical tasks reported have asked participants to estimate their body size by comparing themselves to stimuli which were created by realistically manipulating the BMI dependent body shape changes of the same ‘standard’ 3D model. However, there are two problems with this strategy. The first is that it ignores individual variation in underlying body shape across different participants, as illustrated in Figure 5.1 overleaf. The standard body shape used in the 3D model may have been a good fit for some body shapes, but not so good for others. Therefore, this immediately creates a participant dependent source of variability in the experiment that cannot be controlled easily. Secondly, asking a participant to judge themselves against another individual is one step further removed from what would ideally be the most ecologically valid case – i.e. what we would really like to do is to create a situation equivalent to looking in the mirror. Therefore, we must assume that there is likely to be an additional cognitive load, and therefore an uncontrolled source of variability, in asking participants to map their

belief about their own body shape and size on to a third party. Furthermore, as far as participants with AN are concerned, individual body parts can make a great impact on people's perception of body size. It has been suggested that the overestimation of body size may be due to overestimation of certain body areas (Gardner & Bokenkamp, 1996; Stewart, Klein, Young, Simpson, Lee et al., 2012; Tovée et al., 2003; von Wietersheim, Kunzl, Hoffmann, Glaub, Rottler, 2012), particularly the hips and thighs (Gila, Castro & Toro, 1998; Gila, Castro, Cesena & Toro, 2005).



Figure 5.1: Illustration of real body shape variation, across each row, in individuals with approximately the same BMI.

Other research suggests that individuals with AN avoid looking at the parts of the body they find unsatisfactory in themselves (Jansen, Nederkoorn, & Mulken, 2005; Roefs, Jansen, Moresi, Willems, van Grootel et al., 2008; Smeets, 1999), but may nevertheless be prepared to look at those same areas in other people (Jansen et al. 2005; Roefs et al., 2008). Note however, that with the exception of the experiments reported by Stewart, Klein, Young, Simpson, Lee et al., (2012), these studies have used the Video Distortion Technique (VDT) which, as discussed in the Introduction, can be problematic.

To move towards a solution to these problems, for Experiment 4 we used a Size Stream 3D body scanner to capture a 3D template which accurately represents an individual participant's body shape. This template was then imported to the 3D modelling environment in order to fit the female base model (i.e. Genesis 2) to the template, effectively generating a high resolution clone of an individual's body. Finally, the whole body shape morphs in the modelling environment were added to systematically increase or decrease the BMI of the cloned participant.

We predicted that the pattern of results we found in Experiment 3, Chapter 4, would be repeated in this study for the WHOLE viewing condition in this study, where participants could see the entire avatar and were asked to select the body size they believed themselves to have. In addition, because of studies suggesting that women with AN may avoid viewing body parts that they feel particularly unhappy with, e.g., hips, legs and stomach (Jansen et al., 2005; Slade et al., 1990), and previous body part literature suggesting that individuals with AN overestimate the hip and waist area (Schulze, Scheuerpflug & Buschek, 2008; Skrzypek et al., 2001), we included two restricted viewing conditions: (i) from the waist down (LEG) and (ii) from the waist up (TORSO). We predicted that participants with AN would make

more accurate body size estimates for the TORSO than the LEG condition. Finally, we added an IDEAL viewing condition in which participants could again see the entire avatar, but were asked to select the body size they would like to have. With respect to judgements of ideal body size, Fernández, Probst, Meermann & Vandereycken (1994) used the VDT with 41 eating disordered patients and 31 controls. Every bulimic patient, and 92.6% of their controls expressed the desire to be thinner. In comparison, only 42.9% of the patients with AN wished to be thinner and 23.8% of this group actually wished to be larger. Interestingly, using a different morphing based technique, Tovée et al. (2003) found very similar results.

5.2. Methods

The experimental procedures and methods for participant recruitment for this study were approved by: the local ethics committee at Northumbria University; the Beating Eating Disorders Organisation (BEAT) and the Northern Initiative on Women and Eating (NIWE) Organisation.

5.2.1. Participants

We recruited 30 female participants into the study all of whom have had a formal diagnosis of anorexia nervosa according to DSM-IV-R or DSM-5 (American Psychiatric Association, 2002, 2013). Fifteen of these participants were still receiving treatment at the time of testing, and were assigned to the ‘anorexia nervosa, current’ (ANC) group. The other 15 participants were not currently being treated at the time of testing and were assigned to the ‘anorexia nervosa, history’ (ANH) group. In addition, we recruited 15 female participants from the population of

undergraduate students at Newcastle and Northumbria Universities and from the general population in and around the Newcastle upon Tyne area, all of whom consented to take part in the study as controls (CON). No control participants had a history of eating disorders. Table 5.1 below shows the characteristics of all three participant groups.

Table 5.1: Characteristics of the participants in Experiment 4.

	ANC (<i>n</i> =15)	ANH (<i>n</i> =15)	CON (<i>n</i> =15)	ANC vs ANH	ANC vs CON	ANH vs CON
	<i>M (SD)</i>	<i>M (SD)</i>	<i>M (SD)</i>	<i>P</i>	<i>p</i>	<i>p</i>
Chronological age	24.02 (3.71)	27.60 (8.81)	29.73 (5.31)	.69	.02	.98
BMI	18.44 (2.88)	20.02 (2.60)	23.67 (4.32)	.55	.005	.09
Over-estimation (PSE-BMI)	5.45 (4.74)	0.53 (2.53)	0.27 (2.57)	.01	.01	.78
BSQ	67.53 (16.25)	42.67 (16.83)	51.80 (22.54)	.002	.04	.79
BDI	26.40 (14.95)	12.20 (7.66)	10.13 (9.81)	.02	.01	.99
EDE-Q	3.94 (1.20)	1.77 (1.17)	2.18 (1.29)	<.001	.006	.95
EDE-Q res	3.51 (1.54)	1.57 (1.68)	2.24 (1.43)	.02	.04	.85
EDE-Q wc	3.47 (1.56)	1.07 (1.22)	1.23 (1.35)	<.001	.003	.78
EDE-Q sc	4.09 (1.33)	1.77 (2.65)	2.33 (1.46)	<.001	.01	.88
EDE-Q eat	4.68 (1.05)	2.65 (1.38)	2.90 (1.84)	<.001	.01	.99

Note. ANC: AN participants being treated at the time of testing. ANH: AN participants no longer being treated; CON: healthy, non eating-disordered controls. BMI = Body Mass Index. PSE = Point of Subjective Equality. BSQ = Body Shape Questionnaire. BDI = Beck Depression Inventory. EDE-Q = Eating Disorder Examination Questionnaire global score. EDE-Q res = Eating Disorder Examination Questionnaire eating restraint subscale. EDE-Q wc = Eating Disorder Examination Questionnaire weight concern subscale. EDE-Q sc = Eating Disorder Examination Questionnaire body shape concern subscale. EDE-Q eat = Eating Disorder Examination Questionnaire eating concern subscale.

5.2.2. Psychometric and anthropometric measurements

To assess participants' attitudes towards body shape, weight and eating we used: (i) the 16-item Body Shape Questionnaire (BSQ, range 0-96), (ii) the Eating

Disorders Examination Questionnaire (EDE-Q, range 0-6), (iii) the Beck Depression Inventory (BDI, range 0-63). We also measured participants' body mass index (BMI) with a set of calibrated scales and a stadiometer.

5.2.3. Stimulus image preparation

For the method of constant stimuli (2AFC), see below, we used the CGI image database as reported in Cornelissen et al. (2015). For the method of limits task, see below, CGI avatars were created as follows. First, each participant had a 3D body scan. In a private booth, participants wore underwear only while their body shape was captured using a Size Stream Body Scanner (using scanner software v4.4). This device comprises a set of 14 infra-red depth sensors arranged around the body, each individually fixed to the rigid frame of the booth. Once in the scanner, participants adopted a standard pose while holding hand-rails to steady themselves. They were asked to exhale midway and not to move for ten seconds while the scan was completed. The circumferential inaccuracy of the system, using a test cylinder ~ 880mm tall, is less than +/- 5mm.

The data generated by the scan, a large point cloud, were immediately stored off-line by the Size Stream Studio software, converted into a 30k polygon mesh and this in turn was read into DAZ 3D Studio (v4.8). We used the morphing tools in this modelling environment to restructure the Genesis 2 female base model to have the same shape as the body scan mesh, with the same height, and same bodily proportions. When the modelling was finished, we used the DAZ Measurement Metrics (v1.1) tools to measure the following circumferences from the avatar model: bust, under-bust, waist, hips, upper arm and mid-thigh and compared them to the equivalent measures from the scanned data provided by the Size Stream Scanner

software. Our criteria for an adequate model fit to the scan data were: (i) that these key measurements from Measurement Metrics to be within ± 3 percent of the scan data; (ii) that there should be minimal distance between the scan mesh surface and the model surface throughout the entire model; (iii) that there should be a good qualitative fit between the scan data and the avatar model – i.e. that the avatar should obviously look like body scan mesh. Once these criteria were met, we applied a standard skin to all avatars, a standard sports bikini, and the same background and lighting. We then used the 3Delight render engine (v1.12 bundled with DAZ) to generate a sequence of 120 images from the Genesis 2 avatar in order to represent the body shape changes that occur in the BMI range from ~ 13 to 45.

5.2.4. Psychophysical measurement procedures

Here we used the method of constant stimuli in the two-alternative force choice task (2AFC) to measure each participant's point of subjective equality (PSE), as described in the Methods Chapter 2 and used in Experiment 3. In addition, with the avatar stimuli, participants used the method of limits as described in the Methods Chapter 2, to estimate their body size under each of four task conditions whose order was randomized across participants. To re-iterate, the four viewing conditions and task demands were:

- 1) Viewing the whole avatar (WHOLE); what is my size?
- 2) Viewing the avatar from the waist down to the legs (LEGS); what is my size?
- 3) Viewing the avatar from the waist up to the head (TORSO); what is my size?
- 4) Viewing the whole avatar (IDEAL); what size would I like to be?

5.3. Results

5.3.1. Psychometric task reliability

The responses to the questionnaires across the sample showed good internal reliability scores. For BDI, BSQ and EDE-Q, Cronbach's alpha was: 0.90, 0.96 and 0.90 respectively. For the PSE scores from the 2AFC task, we divided the last block of body size estimation trials into a first and a second half (i.e., in time), in order to estimate split-half reliability. We carried out separate probit analyses on each half of the data to obtain a PSE estimate from each half. The correlation between the two sets of PSE scores ($r=0.98$, $p<.001$) suggests good reliability.

5.3.2. Univariate statistics

Table 5.1 (above) shows the means and standard deviations (SD) for the participant characteristics, separated according to whether they belong to the anorexia nervosa, current (ANC) group, the anorexia nervosa, history (ANH) group or controls (CON). The rightmost columns of Table 1 show the output of pairwise comparisons of these group means, adjusted for multiple comparisons, using the permutation method in PROC MULTTEST (SAS v9.3, SAS Institute, North Carolina, US). Consistent with previous literature we found that when compared to the controls, ANC participants had statistically significantly lower BMIs. In addition, with respect to both the ANH and CON groups, ANC participants had elevated concerns about body shape, eating behaviour and body weight (BSQ, EDE-Q and EDE-Q sub-scores), greater tendency towards depression and significantly greater over-estimation of body size (OE). We found no statistically significant differences between the group means for chronological age.

5.3.3. Whole sample analysis: psychometric measures

Ultimately, we wanted to model the relationships between participants' BMI estimated under the four experimental conditions (i.e. ANC, ANH and CON), using actual BMI and GROUP as predictor variables. In addition, we wanted to control for any influence of AGE and the psychometric variables (BDI, BSQ and EDE-Q). In order to avoid the possibility of introducing substantial variance inflation into the models, we first checked for evidence of co-linearity amongst the psychometric variables. Table 5.2 below shows the Pearson correlation matrix for the psychometric measure across all 45 participants. All correlations were statistically significant at $p < .001$.

Table 5.2: Pearson correlations between psychometric measures from the whole sample in Experiment 4.

	BSQ	BDI	EDE-Q	EDE-Q res	EDE-Q wc	EDE-Q sc	EDE-Q eat
BSQ	-						
BDI	0.64	-					
EDE-Q	0.89	0.65	-				
EDE-Q res	0.65	0.41	0.82	-			
EDE-Q wc	0.79	0.66	0.91	0.63	-		
EDE-Q sc	0.84	0.60	0.94	0.67	0.81	-	
EDE-Q eat	0.92	0.66	0.92	0.62	0.82	0.88	-

Note. BSQ = Body Shape Questionnaire. BDI = Beck Depression Inventory. EDE-Q = Eating Disorder Examination Questionnaire global score. EDE-Q res = Eating Disorder Examination Questionnaire eating restraint subscale. EDE-Q wc = Eating Disorder Examination Questionnaire weight concern subscale. EDE-Q sc = Eating Disorder Examination Questionnaire body shape concern subscale. EDE-Q eat = Eating Disorder Examination Questionnaire eating concern subscale.

Given these substantial correlations, we therefore used PROC FACTOR in SAS v9.3 to carry out an iterated principal factor analysis with rotation in order to identify the significant latent variable(s) in the psychometric data (NB we used the EDE-Q subscores and excluded the global EDE-Q measure, in order to avoid repetition). We then used the factor scores from these latent variable(s) in our statistical models. The

Kaiser-Meyer-Olkin (KMO) measure of sampling adequacy (which indicates the degree of diffusion in the pattern of correlations) was 0.89 suggesting an acceptable sample. One factor had an Eigen value greater than Kaiser's criterion of 1 (i.e. 4.57) which explained 76% of the variance. The scree plot showed an inflexion, i.e. Cattell's criterion which also justified retaining just the one factor. The residuals were all small, and the overall root mean square off-diagonal residual was 0.064, indicating that the factor structure explained most of the correlations. The factor loadings for BDI, BSQ, EDE-Q res, EDE-Q wc, EDE-Q sc and EDE-Q eat were: 0.75, 0.93, 0.76, 0.91, 0.93 and 0.94 respectively. This latent variable, referred to henceforth as PSYCH, represents a combination of the attitudes thought to contribute to body size disturbance: disturbed attitudes to eating, weight, and shape, and depression.

5.3.4. Whole sample analysis: body size over-estimation

Table 5.3 below, shows the Pearson correlations between the psychometric factor PSYCH, chronological age and over-estimation of body size (i.e. estimated BMI minus actual BMI) computed from the four conditions of the method of limits task as well as from the 2AFC method of constant stimuli. The striking finding illustrated in Table 5.3 is that body size over-estimation from the method of limits task, based on viewing either the whole body, or just from waist down, or just from waist up, produced very similar outcomes, all of which are very poorly correlated with judgements about participants' ideal body size. This strongly suggested that we should treat the WHOLE, LEG and TORSO estimates as equivalent to each other [respective means and SEs: 21.88 (0.70), 22.20 (0.75), 21.71 (0.72)], thereby justifying computing an average of these scores per participant, henceforth referred to as AVERAGE. Table 5.3 confirms that the over-estimation of body size computed

from the AVERAGE body size estimate in the method of limits task was also positively and significantly correlated with PSYCH. In addition, PSYCH was not correlated with participants' BMI ($r = -0.04$, $p > .1$), thereby justifying using both BMI and PSYCH as independent explanatory variables in the multivariate analysis. The fact that over-estimation with the method of limits, particularly of the whole body, was well correlated with over-estimation from the 2AFC task (i.e. PSE - BMI), suggests a good degree of similarity between estimates of body size/shape derived from the standard CGI model and individual avatars. Nevertheless, the fact that these correlations are not perfect also suggests that there may be important differences between measurement methods.

Table 5.3: Pearson correlations between PSYCH, chronological age and body size over-estimation across all participants in Experiment 4.

OVER-ESTIMATION	Method of		OVER-ESTIMATION Method of Limits						
			PSYCH	AGE	IDEAL	WHOLE	LEG	TORSO	AVERAGE
			AGE	-	-	-	-	-	-
	Limits	IDEAL	-0.14	-0.36*	-	-	-	-	-
		WHOLE	0.27	-0.19	0.21	-	-	-	-
		LEG	0.40**	-0.08	0.16	0.79***	-	-	-
		TORSO	0.22	-0.15	0.22	0.84***	0.67***	-	-
		AVERAGE	0.33*	-0.15	0.21	0.95***	0.90***	0.91***	-
	2AFC	PSE-BMI	0.42**	-0.33*	0.38*	0.74***	0.59***	0.58***	0.70***

* $p < .05$; ** $p < .01$; *** $p < .001$

5.3.5. Whole sample analysis: comparisons between groups

We used PROC MIXED (SAS v9.3) to build linear mixed models to quantify the relationship between participants' estimates of BMI from their avatars, their actual BMI and GROUP (i.e. ANC, ANH and CON) while controlling for PSYCH and AGE. We ran separate models for AVERAGE BMI estimates (i.e. the average

per participant across the WHOLE, LEG and TORSO conditions) and IDEAL BMI estimates.

The linear mixed model for AVERAGE explained 87% of its variance. All three groups of participants showed a positive, linear relationship between actual BMI and estimated BMI, $F(1,36) = 183.98$, $p < .001$, 95% CI [0.71, 1.22]. Critically, however, the pattern of estimation by women differed between groups because we found a significant main effect of GROUP, $F(2,36) = 3.29$, $p < .05$, 95% CI [ANC -20.28, -2.25; ANH -12.45 6.01] as well as an interaction between GROUP and BMI, $F(2,36) = 4.42$, $p < .05$, 95% CI [ANC 0.19, 1.01; ANH -0.31 0.55]. Neither of the main effects of age, $F(1,36) = 0.74$, $p > .10$, or PSYCH, $F(1,36) = 2.45$, $p > .1$, were statistically significant. Moreover, we found no evidence to justify adding the interaction between PSYCH and GROUP to the model. Given the overall correlation between PSYCH and AVERAGE in Table 5.3, the fact that PSYCH was not significant was somewhat surprising. However, a partial explanation may lie in the fact that computing the correlation between PSYCH and AVERAGE separately for each group, showed an effect restricted to controls only: for ANC, $r = 0.01$, $p > .1$; for ANH, $r = 0.29$, $p > .1$; for CON, $r = 0.57$, $p < .05$. Therefore, any main effect of PSYCH on AVERAGE in the mixed model may simply have been washed out once BMI and AGE were included. Note that the lack of correlation in the two groups of women with a history of AN was not caused by low variability in PSYCH for these two groups compared to controls. The range of z-scores for PSYCH was ANC = -0.70 to 2.20 (2.90), ANH = -1.63 to 0.91 (2.54), CON = -1.56 to 0.99 (2.55).

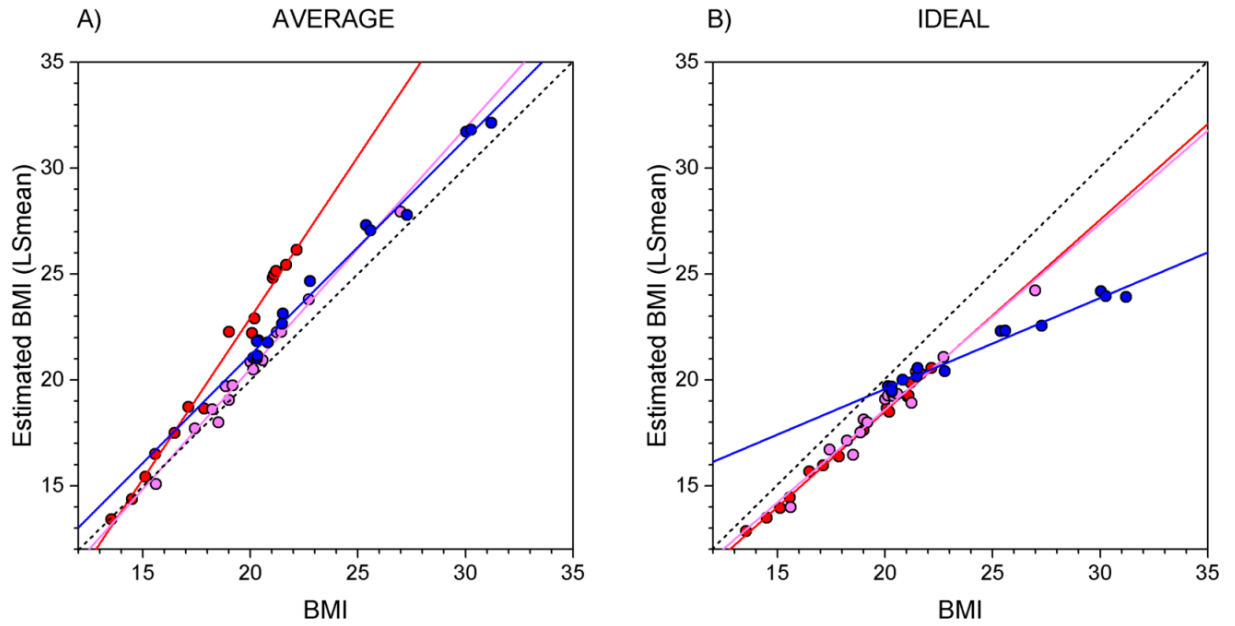


Figure 5.2: Plots of estimated BMI as a function of actual BMI for the data averaged across the belief, leg and torso conditions in A and the ideal condition in B. Red, pink and blue dots with their respective regression lines represent: ANC, ANH and CON participants.

Figure 5.2A illustrates the outcome from the linear mixed model for AVERAGE. The three groups ANC, ANH and CON are represented by: red dots with solid red regression line, pink dots with solid pink regression line and blue dots with solid blue regression line, respectively. The dotted black diagonal line in Figure 5.2 A shows the line of equality (i.e., perfect estimation) between actual BMI (x-axis) and estimated BMI (y-axis) as indexed by the method of limits task. Data points above this line represent over-estimation, while data points below it represent under-estimation.

Table 5.4: Pairwise comparisons of AVERAGE and IDEAL BMI estimates between the ANC, ANH and CON groups of participants, computed for actual BMIs of 15, 25 and 30.

OUTCOME VARIABLE	BMI	Comparison	Differences of LS means	95% CI	<i>p</i>
AVERAGE	15	ANC v ANH	-0.85	-3.76 – 2.04	.55
	15	ANC v CON	-2.26	-5.52 – 1.01	.17
	15	ANH v CON	-1.40	-4.48 – 1.66	.36
	25	ANC v ANH	3.94	0.89 – 6.99	.01
	25	ANC v CON	3.75	1.22 – 6.28	.005
	25	ANH v CON	-0.20	-2.41 – 2.01	.86
	30	ANC v ANH	6.34	1.08 – 11.60	.02
	30	ANC v CON	6.75	2.61 – 10.89	.002
	30	ANH v CON	0.41	-3.64 – 4.46	.84
IDEAL	15	ANC v ANH	-0.46	-3.00 – 2.07	.71
	15	ANC v CON	-3.41	-6.26 – -0.55	.02
	15	ANH v CON	-2.96	-5.63 – -0.26	.03
	25	ANC v ANH	0.12	-2.55 – 2.79	.92
	25	ANC v CON	1.17	-1.04 – 3.39	.29
	25	ANH v CON	1.05	-0.89 – 2.98	.28
	30	ANC v ANH	0.42	-4.18 – 5.02	.85
	30	ANC v CON	3.46	-0.16 – 7.08	.06
	30	ANH v CON	3.04	-0.50 – 6.59	.09

Figure 5.2A therefore shows that both the ANH and CON groups estimated their BMI from their avatars, accurately. This is confirmed by the fact that, for both groups, neither of the regression lines had slopes which were significantly different from 1 ($\beta = 1.1$, $F(1,12) = 0.84$, $p > .1$; $\beta = 1.0$, $F(1,12) = 0.005$, $p > .1$, respectively). By comparison, while low BMI women belonging to the ANC group performed accurately in the task, as this group's actual BMIs increased, so these participants increasingly over-estimated their BMI. This is confirmed by the fact that the slope of the regression of estimated BMI on actual BMI, for the ANC group, was significantly greater than 1 ($\beta = 1.58$, $F(1,12) = 10.59$, $p < .01$). Finally, to confirm the pattern of differences in AVERAGE scores between the three groups, Table 5.4

above, shows pairwise tests between the groups, at three levels of BMI, controlling for multiple comparisons.

The linear mixed model for IDEAL explained 79% of its variance. All three groups of participants showed a positive, linear relationship between actual BMI and estimated BMI, $F(1,36) = 93.01$, $p < .001$, 95% CI [0.24, 0.68]. As with the AVERAGE scores, the pattern of estimation by women differed between groups because we found a significant main effect of GROUP on IDEAL scores, $F(2,36) = 4.27$, $p < .05$, 95% CI [ANC -18.16, -2.39; ANH -17.01 -0.86] as well as an interaction between GROUP and BMI, $F(2,36) = 4.26$, $p < .05$, 95% CI [ANC 0.10 0.82; ANH 0.02 0.77]. Neither of the main effects of age, $F(1,36) = 2.16$, $p > .10$, or PSYCH, $F(1,36) = 0.03$, $p > .1$, were statistically significant.

Figure 5.2B illustrates the outcome from the linear mixed model for IDEAL. As before, the three groups ANC, ANH and CON are represented by: red dots with solid red regression line, pink dots with solid pink regression line and blue dots with solid blue regression line, respectively. The dotted black diagonal line in Figure 5.2B shows the line of equality, i.e., where a participant chooses an avatar whose IDEAL body size is the same as that of the participant. Data points above this line represent a desire for a larger body, while data points below it represent a desire for a smaller body size. Figure 5.2B therefore shows that both the ANC and ANH groups selected ideal body sizes which were lighter than their actual bodies – i.e. equivalent to a fixed reduction in BMI from their actual BMI. Two pieces of evidence suggest this interpretation. First, for both these groups, the slopes of the regressions of ideal avatar BMI on actual BMI were not significantly different from 1 ($\beta = 0.94$, $F(1,12) = 0.11$, $p > .1$; $\beta = 0.98$, $F(1,12) = 0.04$, $p > .1$, respectively). Secondly, as Table 5.5 below shows, the mean difference between IDEAL and BMI for both groups was

negative and significantly less than zero. Indeed, 29/30 participants showed a negative difference, i.e. their IDEAL choice had a lower BMI than their own body. Control participants all chose their IDEAL avatar to have a lower BMI than their actual BMI. However, in this group, the wish to be ‘lighter’ increased systematically with actual BMI, with the greatest reduction for the IDEAL exhibited by those who were the heaviest. This was confirmed by the fact that, for the CON group, the slope of the regression of ideal avatar BMI on actual BMI was significantly less than 1 ($\beta = 0.41$, $F(1,12) = 53.16$, $p < .001$). Again, the pattern of differences in IDEAL scores between the three groups is illustrated by the pairwise comparisons in Table 5.4 above.

Table 5.5: Pattern of differences between the AVERAGE and IDEAL body size estimates and actual BMI in Experiment 4. The p-values represent tests of location against the null hypothesis: $H_0 : \mu_0 = 0$

	AVERAGE - BMI			IDEAL - BMI		
	<i>M</i>	<i>SD</i>	<i>p</i>	<i>M</i>	<i>SD</i>	<i>p</i>
ANC	2.12	2.46	.005	-1.33	1.87	.02
ANH	0.53	1.20	.11	-1.39	1.38	.002
CON	1.01	1.97	.07	-2.86	2.46	.0005

5.4. Discussion

In Experiment 4, body size was estimated by combining the method of limits with avatars of participants built from 3D whole body scan data. Because we wanted participants to estimate their body size under four different task instructions: BELIEF, LEG, TORSO and IDEAL, it would have been too time consuming to use the method of constant stimuli – hence the choice of the method of limits.

The results for the ANC group were very similar to those for participants with ANSD from Experiment 3. The lowest BMI participants in this group accurately estimated their body size. However, as BMI in ANC participants increased, so body size over-estimation occurred in proportion to their BMI. This effect presumably could be attributed to the same explanation that we gave in Experiment 3 – i.e. reducing sensitivity in body size estimation as BMI increases lead to a criterion shift and over-estimation of body size, although we would have needed to be able to measure DL to confirm this. However, the AN participants who were no longer in treatment (i.e. ANH) showed a pattern of results which was indistinguishable from that of healthy controls, when individuals' avatars were used as stimuli instead of the standard model, and for both of these groups the findings were very different from Experiment 3. Specifically, both the control and ANH participants correctly estimated their own body size across the *whole* BMI range; there was no longer any evidence for contraction bias.

The first and most obvious point to make is that, in comparison to Experiment 3 in Chapter 4, obtaining evidence consistent with contraction bias for body size estimation appears to depend on the nature of the task to be used, at least for ANH and healthy control participants. Specifically, for these two groups, when the estimation task required participants to compare themselves to another person, we saw contraction bias (Experiment 3). But when the task required participants to compare themselves to an avatar built from their own 3D body scan, we did not see contraction bias. An important caveat to this observation, however, is that the number of participants in Experiment 4 was smaller than for Experiment 3, owing to the time consuming challenge of building individual avatars. Therefore, it is possible

that had the numbers of participants been larger, and the range of participants' actual BMIs wider, we might have observed exactly the same effects as before.

One very straightforward reason for these differences in the results in the current Experiment 4, comparing to Experiment 3 in Chapter 4, could be the presence of implicit anchoring effects for the method of limits task which were not present for the 2AFC task. Specifically, every trial in the method of limits task started with the presentation of either the very thinnest or the very largest version of each avatar that could be created. Therefore, the two ends of the response range were systematically anchored to the two ends of the stimulus range. Moreover, the stimulus range was effectively the same for all participants. Anchoring in this way is known to at least reduce, if not abolish contraction bias (Poulton, 1989).

In the Introduction and Chapters 3 and 4, the role of a “reference body” for body size estimations was discussed. It was argued that the results in healthy control participants may reflect their need to refer to a distribution of other peoples' body sizes that they have learnt over their lifetimes, in order to judge whether the model they are looking at during the 2AFC task was smaller or larger than they believe themselves to be. In other words, this is exactly the situation that is required in order to observe contraction bias, where reference needs to be made by the observer to a learnt population norm. However, in the current experiment with individual avatars, arguably the body shape being judged was familiar to the participant because it was their own – at least this was certainly the intention. In this case, we speculate that the relevant reference distribution would have been the up-to-date memory of participants seeing their own body in the mirror, in films, photographs and any other source of reflection. These distributions would therefore be normed with respect to each individual's experience, and as a result not be subject to contraction bias, or at

least minimally so. The fact that there was no clear evidence of contraction bias in the participants who were recovering from AN (i.e. ANH), may also be due to these individuals using an up-to-date, self-referential body size distribution in order to make their judgements of BELIEF, LEG and TORSO during the method of limits task. This proposal for CON and ANH participants is illustrated in Figure 5.3 below.

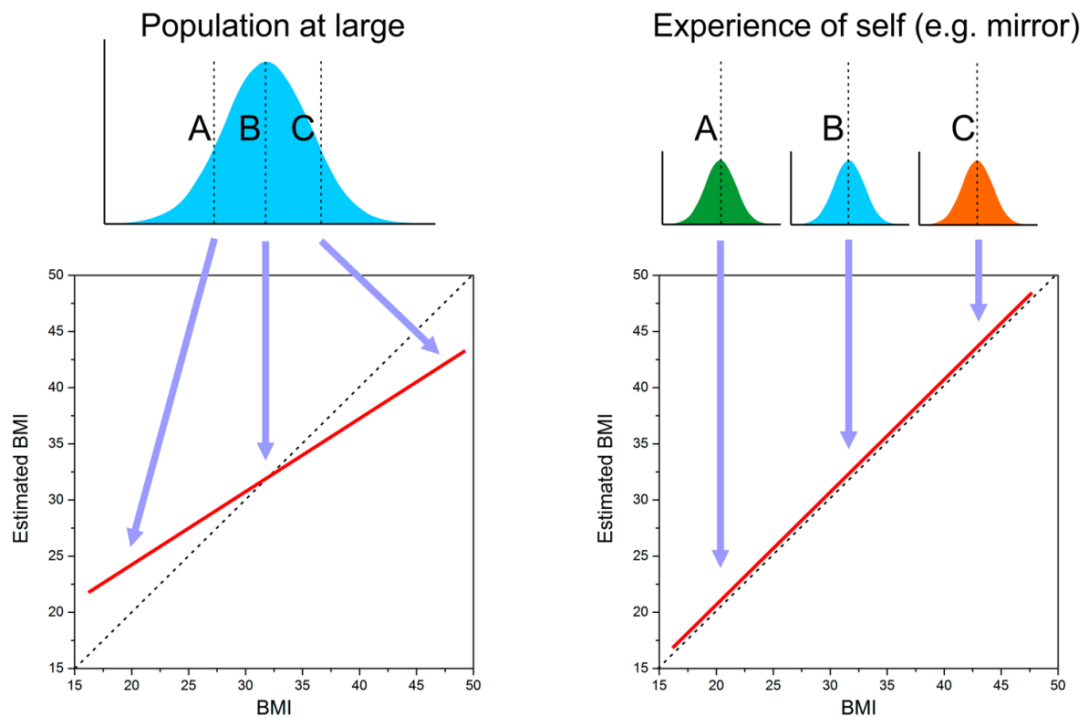


Figure 5.3: Illustration of the proposal that different body size distributions are used by observers as the 'reference' for the 2AFC task (Population at large) and the method of limits task (Experience of self). A, B and C refer to three different individuals with a low, medium and high actual BMI, respectively.

The results from the IDEAL condition were interesting. Here, all but one participant desired an IDEAL body size that had a lower BMI than their own. In the ANC and ANH groups, this was more or less a fixed reduction of about ~ 1.3 BMI

units across the entire BMI range. All of the healthy controls also chose an IDEAL body size with a lower BMI than their own. But in addition, the extent of the desired reduction was linearly related to these individuals' BMI, being greatest in those with the highest BMI. It is hard to understand this dissociation between the performance of those with a history of AN and healthy controls, which was reflected in a significant interaction between BMI and GROUP. However, one speculation might be that women with a history of AN may be seeking a fixed reduction to the body size they currently have, whereas healthy controls might have in mind an external representation of an ideal body shape and weight to which everyone aspires, and which might be heavily culturally biased. For example, Crossley, Cornelissen & Tovée (2012) used similar CGI stimuli to the current experiment to show that women and men choose ideal bodies for themselves that have a lower BMI. With respect to cultural bias, preferences for particular body shapes have been shown to shift as people move between cultures. For example, people living in rural KwaZulu Natal in South Africa prefer a much larger BMI than UK citizens (Tovée et al., 2006). However, people moving to the UK who were originally from KwaZulu Natal shift their preferences towards a lower BMI. The average BMI of people in both regions is not significantly different, but in KwaZulu Natal a heavier body is associated with health and higher socioeconomic status whereas in the UK the opposite is the case.

Stewart et al. (2012) used 3D laser scanning methods, in combination with 2D body shape morphing techniques, to compare body size and shape estimates in 10 participants with anorexia nervosa, 12 with bulimia nervosa and 22 healthy controls. These researchers first confirmed the acceptability of these methods particularly for the patient groups. They also found that all participants wished to be

smaller across all body regions and that patients had poorer veridical perception and greater dissatisfaction than controls. A similar result was reported by Stewart, Crockett, Nevill & Benson (2014) where they extended their methodology to be able to report body shape preference in a tripartite space defined by Sheldon's three somatotypes (Sheldon, Stevens & Tucker, 1940). In short, these authors found that when asked to choose their IDEAL body shape, controls, patients with anorexia nervosa and patients with bulimia nervosa all selected bodies which shifted primarily from endomorphic to ectomorphic (i.e. fat to thin). However, we note that these authors did not explore individual variation in body size / shape estimates with respect to the BMI of the participants involved.

Finally, the effect of PSYCH differs between Experiments 3 and 4. In Experiment 3, when participants made judgements about the standard model during the method of constant stimuli, we found a uniform influence of psychological concerns across the whole sample of participants, irrespective of the group to which they belonged: increasing concerns systematically led to increasing over-estimation. However, for the current Experiment 4, we found no main effect of psychological concerns in the mixed model for AVERAGE. Nevertheless, by computing the correlation between PSYCH and AVERAGE separately for each group, we found a positive correlation for controls, just like Experiment 3, but no significant correlation for women with a history of AN. Therefore, the question seems to be: why do women with a history of AN over-estimate body size as a function of their psychological concerns, when they are judging images of others, but not when judging images of themselves? The answer is not immediately obvious, and will require further research. However, we speculate that a useful conceptual framework to deal with this question may make use of Social Comparison Theory (Festinger,

1954). A tentative working hypothesis might be the following. If we assume that there is a cultural bias towards valuing the thin ideal in women's body shapes (see e.g., Levine & Murnen, 2009), then upward social comparisons (i.e. aspiring to the thin ideal) may lead to the experience of body dissatisfaction, and this may be particularly true for individuals who already have heightened concerns about body shape and weight. Perhaps therefore, when such individuals are asked to pick which version of a standard model they think is closest to their own body size, they are willing to 'pick' an image which is larger than they are because this 'cartoons' their psychological distress. However, at least for women with a history of anorexia, perhaps if they are asked to do the same thing, but from a set of images that they know represent themselves, there is some reason that they either cannot do this, or avoid doing it.

In summary, Experiment 4 replicates our findings from Experiment 3 in respect of women with a history of AN who are still under treatment. However, for women with a history of AN no longer in treatment, as well as for healthy controls, use of the avatar stimuli appeared to remove contraction bias. This result would seem to emphasize that choice of stimulus and task are important if we want participants to estimate body size accurately. Moreover, these findings suggest that the point of view of the participant may also matter (i.e. third person perspective: judging 'another' versus first person perspective: judging 'oneself'), a point we return to in the General Discussion.

Chapter 6

Experiments 5 and 6

Two intervention experiments are described in which an adaptation paradigm was used to measure participants' perceptual boundary for the distinction between "thin" and "fat" images of women. Participants then underwent a four day training period to shift this boundary to a higher level – i.e. where responses to images that would have been classified as "fat", prior to training, were now reclassified as "thin" after training. As a consequence of this perceptual training, reductions in body shape, weight and eating concerns were demonstrated that persisted for up to one month post-training. Experiment 5 tested the effectiveness of the training paradigm in undergraduate students with high body size and shape concerns. Experiment 6 tested the effectiveness of the training paradigm in a sample of women with a history of AN. Both Experiments 5 and 6 were conceived and designed in collaboration with Dr. Martin Tovée and his PhD student Lucinda Gledhill at the Institute of Neuroscience, Newcastle University. The pilot testing for the images to be included in the final set of images for both Experiments 5 and 6 was carried out exclusively by Lucinda Gledhill, and is therefore not reported as a part of this PhD. The data for Experiment 5 were collected by Lucinda Gledhill, and are reported as part of her PhD (Gledhill, 2015). For this PhD, it was felt that a clear and detailed report of Experiment 5 was needed in order to justify Experiment 6. Therefore, with the agreement of Dr Nick Caplan, Director of Postgraduate Research for the Faculty of Health and Life Sciences at Northumbria University, the data from Experiment 5 are treated as a secondary dataset, attributed to Lucinda Gledhill, but analysed de novo for this thesis by Katri Cornelissen. Experiment 6 was carried out and the data analysed exclusively by Katri Cornelissen.

6.1. Introduction

As we have seen in prior literature, and have both confirmed and extended in this thesis, women with AN experience body image distortion such that they tend to over-estimate their body size – the extent depending on their current BMI. Current therapeutic regimes for AN have only a limited success in treating this condition where the long-term mortality rate has been estimated to be as high as 10% (Berkman, Lohr, & Bulik, 2007). The principal treatment for the body image disturbance of AN is cognitive-behavioural therapy (CBT) which tries to modify dysfunctional thoughts, feelings and behaviours that contribute to a negative body image. Other interventions include: fitness training to improve physical capacity and shift attention from appearance to functionality (Farrel, Shafran & Lee, 2006; Jarry & Cash, 2011), media literacy to teach women to challenge thin bodies in the media (Ginis & Bassett, 2011; Martin & Lichtenberger, 2002), self-esteem enhancement to improve self-worth as low self-esteem is associated with negative body image (Grabe, Ward & Hyde, 2008; Irving & Berel, 2001), and psychoeducation addresses issues related to negative body image (O’Dea, 2004. Yager & O’Dea, 2011). However, a recent meta-analysis suggests that once corrections for bias (both within and across studies) in the data were applied, the effect size of these treatments were relatively small and strongly suggest the need for new additional therapies to address negative body image (Alleva et al., 2015).

Therefore, in the current study, we test the effectiveness of a new body training programme which aims to recalibrate the perception of body size in women with AN and so reduce both their body size concerns and their overall eating disordered symptoms. This new therapy for treating body size over-estimation has been developed from a face training program used to modify biases in emotion

recognition in order to encourage the perception of happiness over anger in ambiguous expressions in adolescent youth who are at high risk of criminal offending and delinquency (Penton-Voak, Thomas, Gage, McMurran, McDonald et al., 2013). In the original study, participants were required to categorise faces from a sequence of faces presented individually as either happy or angry (i.e. a 2-alternative forced choice). The faces at the end of the sequence are very clearly either happy or angry, but the faces in the middle of the sequence which were intermediate in their expression could be judged either way. The adolescents at high risk of offending categorised even the intermediate faces as angry. By giving feedback on the accuracy of their judgement, their angry-happy categorical boundary was recalibrated to that of controls. This resulted in a decrease in self-reported anger and aggression and also in independently rated aggressive behaviour. The training has also been used to recalibrate the perception of happiness over anger in ambiguous facial expressions in people suffering depression to improve mood (Adams, Penton-Voak, Harmer, Holmes & Munafò, 2013; Penton-Voak, Bate, Lewis & Munafò, 2012).

To test the potential effectiveness of a modified version of the training regime in treating body image concerns we carried out two experiments. In the first experiment we recruited young women with high concerns about their body size for a randomised control study to determine whether the training program can alter the perceptual position of the thin-fat categorical boundary, and produce an associated improvement in body image and eating concerns. Based on the results from the first experiment, we then applied the training program to a cohort of women with ANSD to determine whether the training would also be effective on their more deep-seated body image concerns.

6.2. Methods

6.2.1. Participants

Participants for this study were recruited from undergraduate students at Newcastle University. Potential participants were all asked whether they had a history of eating disorders and were excluded from further participation if they had. Potential participants were also asked to provide their height and weight and to fill in the 16-item Body Shape Questionnaire BSQ (Evans & Dolan, 1993). This is a 16-item questionnaire that indexes the degree of preoccupation and negative attitude toward body weight and body shape. Only those who achieved a BSQ score of 60 or greater (i.e. individuals with substantial body shape concerns) were eligible to participate in the training study. Accordingly, 20 female participants (mean age = 18.2 years; SD = 0.37) were recruited to the intervention condition, while 20 females (mean age = 19.0 years; SD = 1.3) were recruited to the control condition.

Table 6.1: Summary table of the demographic and questionnaire data from the participants in Experiment 5.

	Intervention (n=20)		Control (n=20)	
	<i>M</i>	<i>SD</i>	<i>M</i>	<i>SD</i>
Age (years)	18.20	0.37	19.00	1.27
BMI	25.40	5.12	23.90	3.01
Screening BSQ	68.20	7.25	73.00	11.52
EDE-Q	3.09	1.01	3.67	0.83
BDI	16.70	9.04	17.80	12.01
RSE	16.00	4.83	13.90	4.45

Note. BMI = Body Mass Index. BSQ = Body Shape Questionnaire. BDI = Beck Depression Inventory. EDE-Q = Eating Disorder Examination Questionnaire global score. BDI = Beck Depression Inventory. RSE = Rosenberg Self Esteem scale.

Table 6.1 above describes the participants' characteristics. Ethical approval for this study was granted by the Faculty of Medical Sciences ethics committee at Newcastle University (00620/2013).

6.2.2. Psychometric and anthropometric measurements

Once recruited, we repeatedly assessed participants' attitudes towards body shape, weight and eating with the Eating Disorders Examination Questionnaire (EDE-Q), and its 4 subscales: (i) the Restraint (EDE-res) subscale investigates the restrictive nature of eating behaviour; (ii) the Eating Concern (EDE-eat) subscale measures preoccupation with food and social eating; (iii) the Shape Concern (EDE-sc) subscale investigates dissatisfaction with body shape and (iv) the Weight Concern (EDE-wc) subscale assesses dissatisfaction with body weight. We also used the Beck Depression Inventory (BDI) that measures participants' level of depression and the Rosenberg Self-Esteem Scale (RSE) that measures self-esteem. In addition, we calculated the participants' body mass index (BMI) from their weight obtained with a set of calibrated scales and their height obtained with a stadiometer.

6.2.3. Stimulus image selection

Based on a series of pilot experiments, all of which were conducted by Lucinda Gledhill at the Institute of Neuroscience, Newcastle University, 15 CGI images were selected that fulfilled two criteria: (i) participants' responses to at least the first and the last images in the sequence (assuming increasing BMI) had to be 100% "thin" and 100% "fat" respectively; (ii) the images needed to increment in BMI in approximately equal steps. The 15 images that were finally selected ranged in BMI from 15.30 to 33.66.

6.2.4. Perceptual training paradigm



Figure 6.1: The middle row shows part of the body sequence varying in BMI from lower to higher. The top row illustrates the results from a baseline assessment and the position of the categorical boundary prior to training. The bottom row illustrates the results from the post-training test session, showing that the categorical boundary has shifted relative to the pre-training result.

We modified an E-Prime (<http://www.pstnet.com/eprime.cfm>) script kindly provided by Ian Penton-Voak [see Penton-Voak et al. (2012, 2013)] to run the training paradigm on a Windows PC with a 19" LCD monitor panel (1600x1200 native pixel resolution, 32-bit colour depth). Each trial of the baseline and training conditions began with a central fixation cross which was shown for 1500-2500 ms (randomly jittered). This was replaced by an image of a body for 150 ms followed immediately by a mask of visual noise which was shown for 150 ms. Finally, the mask was replaced by a prompt screen, containing a "?", to indicate that participants should make their judgement of "fat" or "thin" (i.e. a 2-alternative forced choice) and respond by key-press. No time limits for the response were imposed on the task.

The baseline condition comprised 45 trials in all (i.e. 3 presentations of each of the 15 stimulus images) in randomized order. The software used participants' responses to calculate the categorical boundary/midpoint at which they shifted from perceiving thinness to perceiving fatness in the body sequence 50% of the time (see upper row of Figure 6.1, above).

Trials from the training phase differed from the baseline procedure in that we provided feedback to the participant (i.e. "Incorrect! That body was fat" or "Correct. That body was thin") following their keyboard response. The training phase was made up of 6 blocks, with 31 trials in each block. From pilot testing, bodies 1-2 and 14-15 were almost always classified as 'thin' and 'fat' respectively, so these were only presented once in training. Bodies 3-5 were responded to less frequently as being 'thin', and 11-13 less frequently as being 'fat' and were therefore presented twice. The remaining bodies, 6-10, were presented three times each as responses to these bodies were the least clear-cut.

In the intervention training condition, the nature of the feedback given was 'inflationary' and was designed to shift a participant's categorical boundary by two bodies higher up along the image sequence (from low to high BMI) than the baseline measure. In this way participants were re-trained to judge bodies near their categorical boundary, which they had previously judged as fat during baseline measurement, now to be thin. By contrast, in the control training condition, the feedback to participants was consistent with their categorical boundary as measured at baseline, and was intended merely to reinforce their existing categorical boundary.

6.2.5. Procedure

On Day 1, participants first completed the EDE-Q, RSE and BDI questionnaires and had their BMI measured. Then they carried out the first baseline and training sequences for the categorical perception task. On Days 2 and 3, participants carried out the baseline and the training sequences only. On Day 4, participants completed the baseline and training sequences, followed by the EDE-Q questionnaire. Finally on Day 14, participants carried out the baseline sequence and then completed the EDE-Q questionnaire.

6.3. Results

We used PROC MIXED (SAS v9.3) to fit a multi-level model to the perceptual training data which included three main effects (i.e. group: intervention versus control; training: baseline versus post-training threshold; test day: 1, 2, 3, 4, 14) and all possible two and three-way interactions. In addition, based on significant reductions in $-2\log$ likelihood, we permitted both individual slope and intercept variation for each subject and these were estimated with an ‘unstructured’ variance-covariance matrix. This model allowed us to compute post-hoc pairwise tests, which were controlled for multiple comparisons, as illustrated in Figure 6.2 B and D overleaf. We found statistically significant main effects for test day ($F_{4,304} = 6.69$, $p < .0001$) and training ($F_{1,204} = 15.11$, $p = .0001$) on perceptual thresholds, but not for group ($F_{1,38} = 1.46$, $p = .23$). In addition, we found statistically significant interactions for *group x test day* ($F_{4,304} = 16.62$, $p < .0001$) and *group x training* ($F_{1,304} = 15.93$, $p < .0001$), but not *training x test day* ($F_{3,304} = 0.14$, $p = .93$) nor *training x group x test day* ($F_{3,304} = 0.08$, $p = .97$).

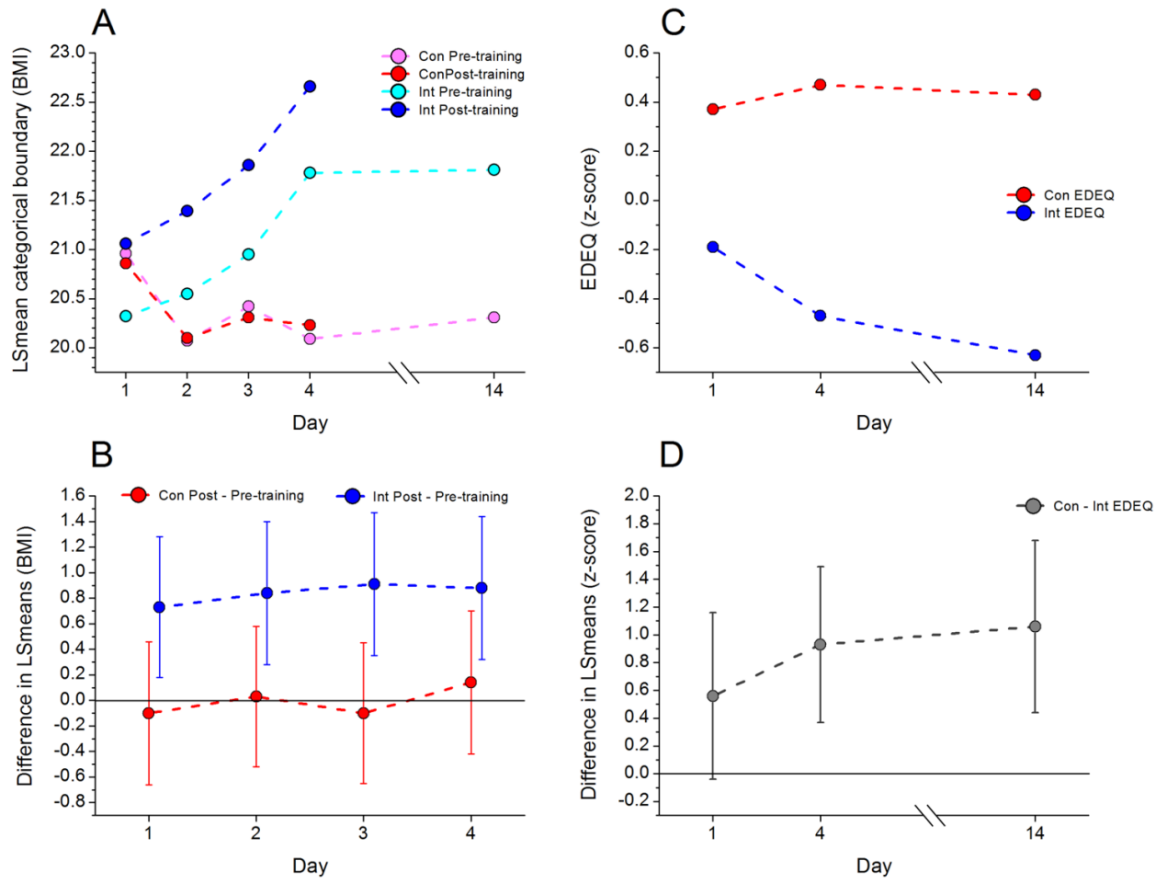


Figure 6.2: A) A plot of the mean value of BMI at the categorical boundary, predicted from the multi-level model as a function of measurement day. Pink and red circles represent control group pre- and post-training thresholds respectively. Cyan and blue circles represent intervention group pre- and post-training thresholds respectively. B) A plot of the predicted differences between pre- and post-training categorical threshold, with 95% CIs, as a function of training day. Confidence intervals that straddle zero are not significant at $p < .05$. Blue circles represent the intervention group and red circles the control group. C) A plot of EDE-Q z-scores as a function of measurement day. Blue circles represent the intervention group and red circles the control group. D) A plot of the predicted differences in EDE-Q between the control and training groups as a function of measurement day, with 95% CIs, as

a function of training day. Confidence intervals that straddle zero are not significant at $p < .05$.

Figure 6.2A shows a plot of the LSmean for the categorical boundary, derived from the multi-level model, as a function of training day. The data are plotted separately for the control (red and pink) and intervention groups (blue and cyan), each split according to whether the measurement was the pretraining baseline (cyan and pink) for that day or the post-training value (red and blue). We found negligible difference between the baseline and post-training thresholds for the controls, as illustrated in Figure 6.2B. This plot shows the LSmean difference between baseline and post-training measurements as a function of training day, separately for the intervention and control groups. The error bars represent the 95% CIs for the pairwise comparisons. By contrast to the controls, the intervention group in Figure 6.2B shows a significant effect of training on each training day. This result, together with the significant *group \times test day* and *group \times training* interactions, shows that training may cause an accumulating shift in participants' categorical boundary towards heavier bodies for the intervention group, but not the control group. Finally, we found that the comparison between Day1 baseline and Day 14 baseline showed a statistically significant increase in BMI at the category boundary for the intervention group ($t_{53.7} = 4.42$, $p < .0001$). In comparison, the controls showed a small reduction that was not statistically significant ($t_{53.7} = -1.86$, $p = .07$). This suggests that, in this sample of non-eating disordered participants, all of whom have high body shape concerns, the perceptual training effect on their categorical boundaries persisted in the intervention group for at least two weeks.

Table 6.2: Summary table of the psychological outcomes from Experiment 5.

Measure	Test day	Intervention	Control	Difference	95% CI	p-value
EDE-Q	1	-0.19	0.37	0.56	-0.01 – 1.13	.06
	4	-0.47	0.47	0.93	0.37 – 1.49	.002
	14	-0.63	0.43	1.04	0.44 – 1.68	.001
EDE-res	1	-0.22	0.36	0.58	-0.04 – 1.21	.07
	4	-0.40	0.39	0.79	0.20 – 1.38	.01
	14	-0.53	0.39	0.92	0.33 – 1.51	.003
EDE-eat	1	-0.08	0.42	0.50	-0.18 – 1.18	.14
	4	-0.34	0.29	0.62	0.09 – 1.16	.12
	14	-0.42	0.13	0.55	-0.09 – 1.19	.09
EDE-sc	1	-0.25	0.38	0.63	0.03 – 1.23	.04
	4	-0.37	0.45	0.81	0.24 – 1.38	.006
	14	-0.62	0.42	1.04	0.45 – 1.64	.001
EDE-wc	1	-0.07	0.02	0.09	-0.50 – 0.68	.75
	4	-0.46	0.51	0.97	0.37 – 1.57	.002
	14	-0.57	0.58	1.15	0.58 – 1.72	.0002

For the psychological measures, we used PROC MIXED (SAS v9.3) to fit separate multi-level models to participants' EDE-Q, EDE-res, EDE-eat, EDE-sc and EDE-wc scores, measured on: Day 1 before the perceptual training, Day 4 after perceptual training and on Day 14 after the baseline perceptual measurement. The psychological measures were transformed in to z-scores to facilitate comparison between them. Each model contained group and test day as main effects, together with the interaction *group x test day*. As before, based on significant reductions in -2log likelihood, we permitted both individual slope and intercept for each subject and estimated these with an 'unstructured' variance-covariance matrix. The post-hoc pairwise comparisons are all reported in Table 6.2 above, and the data for EDE-Q are plotted in Figure 6.2 C and D.

Table 6.2 shows that, with the exception of the EDE-eat scores, we found statistically significant reductions in EDE-Q, EDE-res, EDE-wc and EDE-sc scores

for the intervention group compared to controls for test days 4 and 14 but not day 1. Broadly, these results therefore suggest a pattern whereby the visual perceptual training effect may also have a beneficial influence on participants' attitudes to body shape, weight and some aspects of their eating behaviour. Finally, when Day1 scores were compared with Day 14 scores on the EDE-Q and its subscales, the EDE-Q global score showed a modest, albeit marginally significant, reduction for the intervention group ($t_{73.5} = -1.81$, $p = .07$), and no difference for controls ($t_{73.5} = 0.24$, $p = .81$). The EDE-wc subscale of the EDE-Q showed a significant reduction for the intervention group ($t_{73.3} = -1.94$, $p = .05$), and a significant increase for controls ($t_{73.3} = 2.16$, $p = .03$). Put together, these results suggest that, in a non-eating disordered sample of participants, all of whom have high concerns about body shape, the perceptual intervention may shift attitudes to body shape, weight and eating in a way that would be useful in patients with eating disorders.

6.4. Discussion

Experiment 5 demonstrated that the training program is able to significantly shift the thin-fat categorical boundary in individuals with high body concerns. This change is specific to the intervention group. There was a significant reduction in the EDE-Q scores, particularly on the restraint, weight and shape sub-scales, suggesting that the training not only shifts categorical boundary but also generalises to impact on body size and shape concerns and eating restraint. These changes were retained at 2 weeks post training, suggesting that these may represent statistically significant and long lasting changes to attitudes to body size and eating. This suggests that the training program has the potential to be used to improve body image. However, it can be argued that women with an eating disorder have more deep seated concerns

that may be harder to modify. To test the training's effectiveness and feasibility we therefore recruited from an eating disordered population for Experiment 6.

6.5. Experiment 6: Rationale

In this experiment, we recruited an eating disordered sample of women to determine whether it was also possible to alter their thin-fat categorical boundary and whether there would be an associated positive change in their mental state. In the absence of a control group, we felt that we should control for the possibility that changes in the psychological scores over time might occur merely as a result of habituation to the task, given that the EDE-Q was repeated over multiple test days. For this reason, we included another cognitive task, the Wechsler Adult Intelligence Scale (WAIS-II) Digit Span, the responses to which should not be influenced by concerns about body shape and weight. Nevertheless, because we administered this task as many times as the psychological questionnaires about body shape, weight and eating, we should also expect there to be changes in participants' responses which are related to practice effects. Therefore, we included Digit Span as a covariate in our analyses of the psychological data, on the grounds that it should control for such influences, and any real changes in attitude towards body image should survive this statistical control.

6.6. Methods

6.6.1. Participants

We recruited 21 female participants with ANSD into the study. Three ANSD participants failed to adhere to the training regime, therefore we ran two different analyses. In the first, based on an intention to treat, we included all 21 participants.

In the second analysis, we excluded these three participants. Table 6.3 describes the participant characteristics for all 21 individuals.

Table 6.3: Summary table of the demographic and questionnaire data from the participants in Experiment 6.

	ANSD (n=21)	
	<i>M</i>	<i>SD</i>
Age (years)	26.8	6.32
BMI	19.1	2.76
BSQ	60.4	19.36
EDE-Q	3.4	1.66
DIGIT SPAN	13.7	3.48

The experimental procedures and methods for participant recruitment for Experiment 6 were approved by: the local ethics committee at Northumbria University; the Beating Eating Disorders Organisation (BEAT) and the Northern Initiative on Women and Eating (NIWE) Organisation.

6.6.2. Psychometric and anthropometric measurements

We used the same psychometric, anthropometric and psychophysical measures as in Experiment 5. However, we did not include the BDI and RSE because Experiment 6 included one more measurement point than Experiment 5, and we did not want to over-burden participants. In addition, as mentioned above, we included the Digit Span task from the WAIS-R IQ test battery (Wechsler, 1981) which assesses short term memory.

6.6.3. Procedure

On Day 1, participants first completed the Digit Span task, the BSQ and EDE-Q questionnaires and had their BMI measured. They then carried out the first baseline and training sequences for the categorical perception task. On Days 2 and 3, participants carried out the baseline and the training sequences only. On Day 4, participants completed the Digit Span task and the EDE-Q questionnaire, followed by the baseline and training sequences. On Days 7 and 30, participants completed the Digit Span task and the EDE-Q questionnaire and carried out the baseline sequence only.

6.7. Results

Based on intention to treat, we used PROC MIXED (SAS v9.3) to fit a multi-level model to the perceptual training data for all 21 participants. This included two main effects (i.e. training: baseline versus post-training threshold; test day: 1, 2, 3, 4, 7, 30) and the two-way interaction between them. Based on significant reductions in -2log likelihood, we permitted both individual slope and intercept variation for each subject and these were estimated with an ‘unstructured’ variance-covariance matrix. As before, this allowed us to compute post-hoc pairwise tests, which were controlled for multiple comparisons. These are illustrated in Figure 6.3B&D below. We found statistically significant main effects of test day ($F_{5,16.6} = 4.07$, $p = .01$) and training ($F_{1,64} = 21.53$, $p < .0001$) on perceptual thresholds, but no significant interaction between them ($F_{3,64} = 1.13$, $p = .35$).

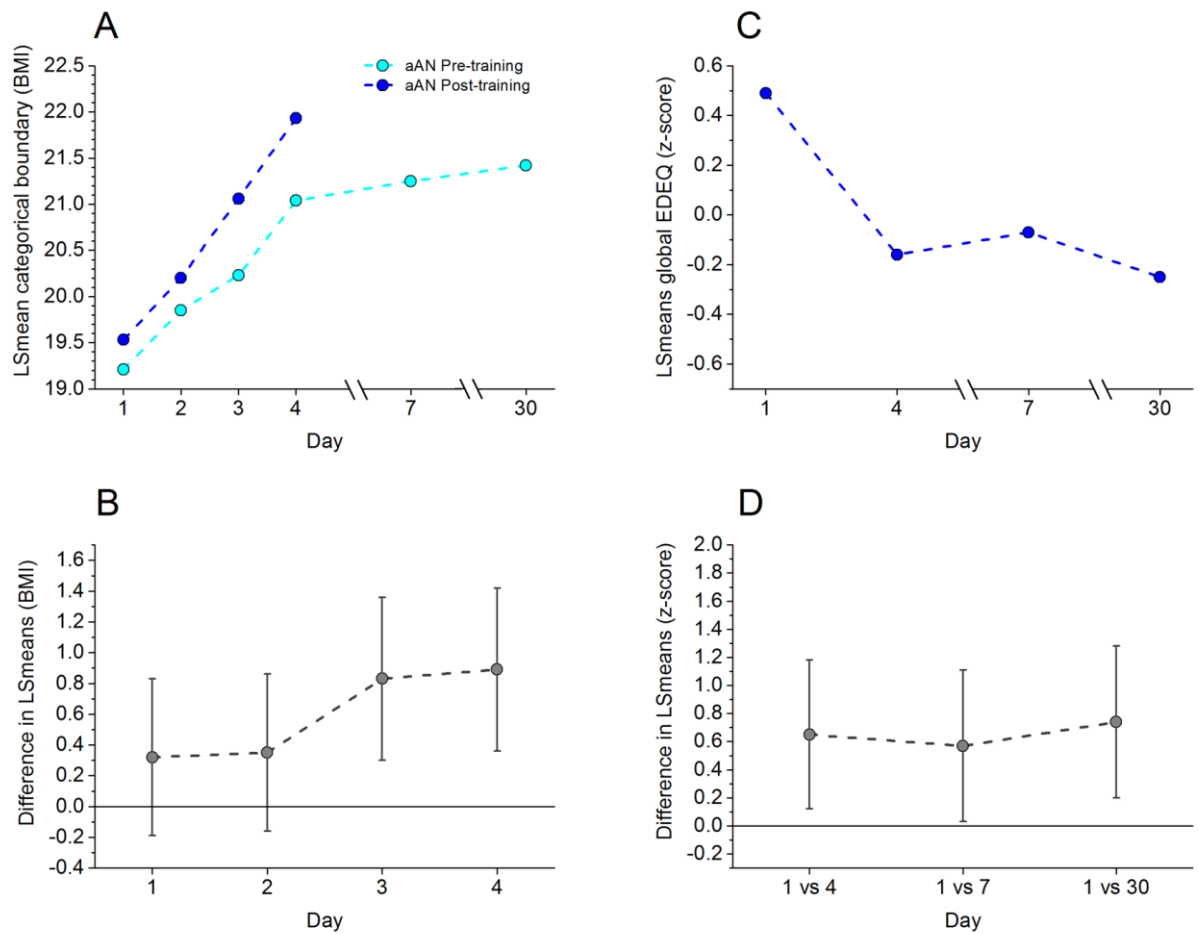


Figure 6.3: A) A plot of the mean value of BMI at the categorical boundary, predicted from the multi-level model as a function of measurement day. Cyan and blue circles represent pre- and post-training thresholds respectively for the ANSD participants in experiment 6. B) A plot of the predicted differences between pre- and post-training categorical threshold, with 95% CIs, as a function of training day. Confidence intervals that straddle zero are not significant at $p < .05$. C) A plot of EDE-Q z-scores as a function of measurement day for ANSD participants in experiment 6. D) A plot of the predicted differences in EDE-Q between the baseline measurement on day 1 and the post-training measurements on days 4, 7 and 30, with 95% CIs. Confidence intervals that straddle zero are not significant at $p < .05$.

Figure 6.3A above, shows a plot of the LSmean categorical boundary from the perceptual task, derived from the multi-level model, as a function of training day. The data are plotted separately for the pretraining baseline for that day (cyan) or the post-training value (blue). Statistically significant differences were found between the baseline and post-training thresholds for days 3 and 4, but not 1 and 2, as illustrated in Figure 6.3B. Figure 6.3A shows a somewhat surprising difference between the Day 1 post-training threshold (19.53) and a *higher* Day 2 pre-training threshold (19.85). However, this difference was not statistically significant ($t_{38.8} = 0.95$, $p = .348$). Overall, therefore, these results, together with the significant main effects of test day and group, show that training may cause an accumulating shift in the categorical boundary towards heavier bodies in the ANSD participants. Finally, the comparison between Day1 baseline and Day 30 baseline showed a statistically significant increase in categorical boundary ($t_{21.9} = 3.06$, $p = .006$). This suggests that, in this sample of ANSD participants, the perceptual training effect persisted for at least a month.

For the psychological measures, we used PROC MIXED (SAS v9.3) to fit separate multi-level models to participants' EDE-Q, EDE-res, EDE-eat, EDE-sc and EDE-wc scores, measured on Days 1, 4, 7 and 30. As in Experiment 5, we transformed the psychological measures in to z-scores to facilitate comparison between them. Each model comprised the main effect of test day, together with chronological age and Digit Span as covariates. As in Experiment 5, based on significant reductions in -2log likelihood, we permitted both individual slope and intercept variation for each subject and these were estimated using an 'unstructured' variance-covariance matrix. The post-hoc pairwise comparisons are all reported in Table 6.4, and the data for EDE-Q are plotted in Figure 6.3 C&D.

Table 6.4 below, shows that, in the smaller sample of 18 ANSD participants who completed the training regime as prescribed, perceptual training was associated with significant reductions in body shape, weight and eating concerns (with the exception of EDE-res), even when Digit Span was controlled for. Moreover, these reductions persisted up to a month from initial testing.

Table 6.4: Summary table of the psychological outcomes from Experiment 6.

Measure	Test day	z-score	Comparison Point	Difference	95% CI	p-value
EDE-Q	1	0.49	Day 1 vs 4	0.65	0.13 – 1.18	.02
	4	-0.16	Day 1 vs 7	0.57	0.02 – 1.12	.04
	7	-0.07	Day 1 vs 30	0.74	0.20 – 1.28	.008
	30	-0.25				
EDE-res	1	0.48	Day 1 vs 4	0.59	-0.07 – 1.25	.08
	4	-0.11	Day 1 vs 7	0.65	-0.02 – 1.32	.06
	7	-0.17	Day 1 vs 30	0.66	-0.004 – 1.33	.05
	30	-0.19				
EDE-eat	1	0.57	Day 1 vs 4	0.79	0.14 – 1.43	.02
	4	-0.21	Day 1 vs 7	0.66	-0.03 – 1.35	.06
	7	-0.10	Day 1 vs 30	0.84	0.22 – 1.47	.01
	30	-0.27				
EDE-sc	1	0.52	Day 1 vs 4	0.73	0.14 – 1.32	.02
	4	-0.21	Day 1 vs 7	0.59	-0.06 – 1.25	.07
	7	-0.07	Day 1 vs 30	0.76	0.15 – 1.36	.01
	30	-0.24				
EDE-wc	1	0.31	Day 1 vs 4	0.38	-0.15 – 0.92	.16
	4	-0.07	Day 1 vs 7	0.33	-0.23 – 0.89	.25
	7	-0.01	Day 1 vs 30	0.54	0.001 – 1.09	.05
	30	-0.23				

6.8. Discussion

Previous research has shown that body size can be judged in a categorical manner, such that a perceptual boundary between “thin” and “fat” body sizes can be

estimated for individual participants (Tovée et al., 2012). In this set of experiments, we tested whether a cognitive bias modification technique, adapted from Penton-Voak et al. (2012; 2013), could shift this perceptual boundary towards larger bodies, and if so, whether this might reduce body image concerns in participants with AN.

In Experiment 5, we recruited young women with high body size concerns (but without a formal diagnosis of AN) to test whether their thin-fat categorical boundary could be altered. The results showed a significant shift in the categorical boundary in the intervention group relative to the controls, and this shift was retained 2 weeks after the training. The EDE-Q scores (a general measure of eating disordered concerns which is often used as a screening questionnaire) showed a significant reduction in the intervention condition relative to controls, and this reduction persisted 2 weeks after the end of training.

In Experiment 6, we tested the intervention on a cohort of women with ANSD. The follow up period was also extended from 2 weeks to a month to test retention of the training. This second experiment did not have a control group, but we attempted to compensate for this, in part, by using a digit span task as a control task. As this task is unrelated to body image and eating disorders and should be unaffected by the body training program, it can act as a proxy for any potential practice effects due to the fact that the EDE-Q was repeated across measurement time points. As in Experiment 5, we found a significant shift in the categorical boundary during training which was retained a month later. This was accompanied by a significant reduction in the EDE-Q scores over the course of training and which was also retained a month later. This change was still statistically significant even after accounting for practice effects by including digit span as a covariate in the analysis. What is important here is not that women with ANSD can be trained to

alter the categorical boundary at which they classify a body to be “fat”, but that this change is retained and seems to generalise to other aspects of their body image and eating disordered concerns.

A key question is whether the reductions that we observe in psychological concerns over a month in the ANSD participants might be clinically meaningful. With respect to EDE-Q, Bardone-Cone, Harney, Maldonado, Lawson, Robinson et al. (2010) operationalize recovery in eating disordered patients as a reduction in all four sub-scale scores to within 1 *SD* of age-matched community norms. Mond et al. (2006) report such norms for the age group 23 to 27 years based on a sample of 908 women: EDE-Q $M = 1.56$, $SD = 1.26$; EDE-res $M = 1.34$, $SD = 1.39$; EDE-eat $M = 0.81$, $SD = 1.10$; EDE-sc $M = 1.84$, $SD = 1.50$; EDE-wc $M = 2.24$, $SD = 1.61$. The mean EDE-Q and sub-scale scores from our ANSD sample (mean age 26.8 years) at Day 30, were respectively: EDE-Q $M = 2.63$; EDE-res $M = 2.45$; EDE-eat $M = 2.06$; EDE-sc $M = 2.83$; EDE-wc $M = 3.18$. Therefore, with the exception of EDE-eat, which missed the criterion by only 0.15 units, the perceptual training regime reported here produced reductions in EDE-Q scores which were clinically meaningful, at least when defined in this way.

Current treatments focus mainly on the cognitive component of the body image distortion, with many failing to recognise the importance of perceptual body image distortion (see, e.g., Hay et al., 2015). The two experiments reported here suggest a promising new way of treating biases in the judgement of female body size which is characteristic of AN. This therefore has the potential to provide a cost-effective adjunctive treatment for AN which may be used together with more traditional talking therapies (cognitive behavioural therapy, mindfulness etc.).

In conclusion, the evidence from these two experiments suggests that perception training can be useful in resetting the perceptual boundary for thin/fat body size and reducing body dissatisfaction, thus reducing the body image distortion in eating disordered individuals.

Chapter 7

Experiment 7

As we have seen, most studies have found that patients with AN overestimate their body size when compared to healthy controls. Moreover, women with AN have a different pattern of eye-movements compared to healthy controls, when judging bodies. However, it is unclear whether this is specific to their diagnosis or whether it is found in anyone over-estimating body size. To address this question, we compared the eye movement patterns from three participant groups while they carried out a body size estimation task: (i) 20 women with recovering/recovered anorexia, ANSD, who had concerns about body shape and weight and who over-estimated body size, (ii) 20 healthy controls who did not have concerns about body shape and who estimated body size accurately, CN(ACC) (iii) 20 healthy controls who did not have concerns about body shape but who did over-estimate body size, CN(OVER).

7.1. Introduction

Although human eyes can attend to a binocular visual field of around 160° , detailed information can only be received from a central region of around 2° , corresponding to the fovea (Levi, Klein & Aitsebaomo, 1985). As a result, the information in an image can only be sampled in discrete “bite-sized” chunks corresponding to the observer’s current fixation position (e.g., Yarbus, 1967). Thus, the areas of the body that are fixated during a body size estimation form the basis of the visual information used to make this judgement. The shape of the body differs at different BMI levels, and some parts of the body change in size and shape more than

others as BMI changes (i.e. the shape and size of some parts of the body are potentially better cues to BMI than others) (e.g., Cornelissen, Hancock, Kiviniemi, George & Tovée, 2009a; Crossley et al., 2012; Wells et al., 2007). For example, stomach depth is very highly correlated with BMI and so could be a good cue to use when judging body weight (Cornelissen et al., 2009b). Fixating the areas which are sensitive to changing BMI is potentially a good strategy for sampling reliable visual cues to judge BMI. Therefore, a less efficient strategy which samples areas of the body that are less sensitive to weight change could lead to a mis-estimation of body weight.

A number of studies have compared fixation patterns between women with AN (or women at high risk of eating disorders) and healthy controls. However, although they report differences in the fixation patterns between the women with AN and controls, there is only limited consensus on which areas are favoured or avoided (Cho & Lee, 2013; George, Cornelissen, Hancock, Kiviniemi & Tovée, 2011; Hewig, Cooper, Trippe, Hecht, Straube et al., 2008; Horndasch, Kratz, Holcinger, Heinrich, Hönig, Nöth et al., 2012; Janelle, Hausenblas, Ellis, Coombes & Duley, 2009; von Wietersheim, Kunzl, Hoffmann, Glaub, Rottler et al., 2012). This may be due to methodological differences. For example, there is considerable variability in the stimuli used; in their pose, whether the images were in colour or black and white, whether the face or head was shown in the image, the length of the presentation, whether the pictures were of real bodies or of computer generated bodies, and in the type and amount of clothing they were wearing (Cho et al., 2013; George et al., 2011; Hewig et al., 2008; Horndasch et al., 2012; Janelle et al., 2009; von Wietersheim et al., 2012). Perhaps the most important factor in these differences is the choice of the free-viewing condition for stimulus presentation (i.e. the

participants look at bodies without any specific judgement being required of them) (Cho et al., 2013; Hewig et al., 2008; Horndasch et al., 2012; von Wietersheim et al., 2012). Instead, the eye-movements are related to the participants' scores on psychometric measures (Cho et al., 2013; Hewig et al., 2008; Horndasch et al., 2012) or to behavioural judgements which have been made at a different time and not during eye-movement recording (von Wietersheim et al., 2012). This is important because the pattern of fixations is significantly different for different judgements, such as between judgements of attractiveness, body size and body shape (Cornelissen et al., 2009a). In a free viewing condition, it is not clear what judgement is being made by a participant. Therefore in this study, we recorded eye-movements from our participants while they were explicitly making a body size judgement so that we could measure which parts of the body were fixated during that discrimination.

In the current study, the aim was to separate out eye-movement patterns that may be specifically associated with the accuracy of body size judgements from those that may be specific to having a clinical diagnosis of AN, including any associated neurological or eye movement control deficits as well as attitudinal concerns over body size and shape. To do this, a careful selection of participant groups was necessary. The participant groups chosen were: (i) women with AN who have concerns about body shape and weight and who do over-estimate body size when tested behaviourally, (ii) healthy controls who do *not* have concerns about body shape but who do over-estimate body size behaviourally, i.e. CN(OVER), (iii) healthy controls who do *not* have concerns about body shape and who do *not* over-estimate body size, i.e. CN(ACC). A simple comparison between women with AN and CN(ACC) may reveal differences in fixations that produce an over-estimation in

body size estimation or they may be specific to this eating disordered diagnosis. However, if by adding the comparison between AN and CN(OVER) we see the same pattern of differences, these secondary factors can be discounted.

An important additional constraint for the design of this study was the requirement that all the participant groups should have the same average BMI. We adopted this strategy in order to prevent the perceptual biases identified in Chapters 3 and 4, which depend on an observer's BMI, having any influence on participants' behavioural decisions or gaze patterns. To achieve this, (cf. Chapter 4), we recruited participants with a history of AN and who had as wide a range of BMI as possible. This meant that the eating disordered group, referred to as ANSD, no longer fitted the DSM-5 diagnostic criteria with respect to BMI, although they still have very high body size concerns and eating disordered behaviours.

7.2. Methods

The experimental procedures and methods for participant recruitment for this study were approved by: the local ethics committee at Northumbria University; the Beating Eating Disorders Organisation (BEAT) and the Northern Initiative on Women and Eating (NIWE) Organisation.

7.2.1. Participants

We adopted the same strategy for recruiting participants to this study as in Experiment 3, Chapter 4. Participants were recruited from the population of staff and students at Newcastle and Northumbria University and from the general population in and around Newcastle upon Tyne. Potential control participants for the current study were selected initially only on the basis that they had no history of eating

disorders. We did not apply other psychometric constraints because we wanted to recruit individuals who are representative of the non-eating disordered female population, many of whom have concerns about body image (e.g., Mond et al., 2006). We then ran the same 2AFC body size estimation task as we used in Experiment 3 to measure PSE and DL. From the PSE scores, we identified whether control participants would be assigned to the over-estimating i.e. CN(OVER) or the accurately estimating i.e. CN(ACC) control group. The criteria for accurate estimation versus overestimation of body size was a difference between the participant's self-estimate from psychophysical testing and their actual BMI no greater than ± 0.7 BMI units versus greater than $+1$ BMI unit, respectively. These criteria were based, in turn, on the psychophysical estimates of the smallest just noticeable difference for BMI (i.e. ~ 1 BMI unit) that we obtained in Experiment 2a, Chapter 3. Control participants were recruited in to each group until we had 20 CN(ACC) and 20 CN(OVER) participants.

Again, very much like Experiment 3, Chapter 4, participants for the ANSD group were recruited if: (i) they had a formal diagnosis of anorexia nervosa according to DSM-IV-R or DSM-5 or bulimia nervosa followed by the onset of anorexia. Note that for the current study we relaxed any BMI criteria, to ensure comparability with the controls; (ii) they over-estimated their own BMI by at least 1 BMI unit. Ten of our 20 ANSD participants were being treated at the time of testing, while 10 were no longer receiving treatment. We used the permutation method in PROC MULTTEST (SAS v9.3) to compute pairwise comparisons between these two subgroups of ANSD participants, adjusted for multiple comparisons. There were no statistically significant differences between the two sub-groups of ANSD participants for chronological age, BMI, BDI, EAT, BSQ, EDE-Q, RSE, PSE and

DL. Therefore, henceforth, the ANSD group was treated as a single group of individuals for purposes of comparison with the two control groups. Finally, there were no statistically significant differences in BMI between the ANSD, CN(ACC) and CN(OVER) groups of participants. Table 7.1 details participant characteristics.

Table 7.1: Participant characteristics for Experiment 7

Variable	ANSD	CN (ACC)	CN (OVER)	ANSD v CN (ACC)	ANSD v CN (OVER)	CN (ACC) v CN (OVER)
	<i>M (SD)</i>	<i>M (SD)</i>	<i>M (SD)</i>	<i>p</i>	<i>p</i>	<i>p</i>
Age	23.70 (4.43)	23.25 (7.93)	20.60 (2.89)	ns	ns	ns
BMI	21.71 (3.95)	23.01 (4.11)	23.19 (5.10)	ns	ns	ns
BDI	26.06 (10.18)	10.47 (6.00)	11.05 (7.22)	< .001	< .001	ns
EAT	32.68 (15.82)	14.30 (9.37)	11.55 (9.08)	< .001	< .001	ns
BSQ	70.79 (13.62)	54.45 (18.20)	52.00 (15.02)	< .05	< .005	ns
EDE-Q	3.67 (1.40)	2.25 (1.33)	1.96 (1.05)	< .05	< .001	ns
RSE	11.42 (4.41)	17.53 (4.0)	16.88 (4.83)	< .001	< .005	ns
PSE-BMI	3.94 (1.96)	-0.07 (0.46)	3.01 (1.19)	< .001	ns	< .001
DL	0.87 (0.81)	0.74 (0.37)	1.13 (0.78)	ns	ns	ns

Note. BMI = Body Mass Index. BDI = Beck Depression Inventory. EAT = Eating Attitudes Test. BSQ = Body Shape Questionnaire. EDE-Q = Eating Disorder Examination Questionnaire global score. RSE = Rosenberg Self-Esteem Scale. PSE = Point of Subjective Equality. DL = Difference Limen.

In summary, our experimental design required, and Table 7.1 confirms that (i) participants with recovering/recovered anorexia nervosa (ANSD) had the same average BMI as the two control groups; (ii) that the attitudes of ANSD participants towards body shape, weight and eating were substantially impaired compared to controls; (iii) only body size estimation discriminated between the two control groups.

7.2.2. Psychophysical measurements

As has already been stated, we used the same 2AFC method of constant stimuli task as in Experiment 3, Chapter 4, to identify whether potential participants estimated body size accurately, or whether they over-estimated.

7.2.3. Eye movement recordings

In the eye-movement task, the participants completed an abbreviated version of the 2AFC psychophysical procedure to estimate body size while their eye-movements were recorded. Participants sat in a dimly illuminated room with their heads supported by a combined head and chin rest. We recorded movements of their right eye with an Eyelink 1000 eye-tracker at a sample rate of 1000Hz. We presented stimuli on a cathode ray tube (CRT) monitor at a standard viewing distance of about ~60cm. At the start of the session, participants' eye movements were calibrated using a 9 point calibration screen. Once the calibration procedure was validated, the experimental task began. We presented eleven stimuli, selected from the same database of 3D rendered images used for psychophysical assessment in Chapter 4, Experiment 3. These covered the BMI range from ~12 to ~45 in equal steps. The experimental task comprised 110 trials (i.e. 11 stimulus images each presented 10 times, in random order). The presentation of each body was preceded by a fixation spot, picked at random from one of four locations: top left, bottom left, top right or bottom right of the presentation screen. When the eye-tracker software had detected that the participant had continuously fixated the fixation spot for 1000ms, it was replaced by one of the 11 stimuli which appeared centred on the screen. The requirement to fixate the fixation spot prevented any anticipatory eye movements. The stimulus remained on screen for as long as it took the participant to decide whether the woman in the image was smaller or larger than they believed themselves to be. The trial ended when the participant pressed the appropriate button to indicate their decision and the next trial was initiated. Behavioural responses from the eye movement recording sessions were treated in a similar fashion to the 2AFC task – i.e. obtaining each participant's psychometric function by probit analysis off-line.

Note however, that these estimates of PSE are necessarily less refined because they are based only on 11 stimuli covering the full BMI range from ~12 to ~45.

The Eyelink 1000 system uses a saccade-picker approach to identify saccades by applying an exclusive OR rule to three thresholds: velocity (30 degrees/sec), acceleration (8000 deg/sec²) and distance moved between samples (0.1deg). It then treats the rest of the (non-blink) data as fixations, assuming that the ‘not in a saccade’ condition is maintained for at least 50ms. The stated accuracy of the system is down to a resolution of 0.15°, though 0.25° to 0.5° is typical.

7.2.4. Eye movement analysis path

As with many eye movement studies, we wanted to analyse the data in such a way as to reveal whether and where any differences in fixation patterns might exist between the three experimental groups. In the literature, a common way to achieve this has been to predefine areas of interest (AOI) on stimulus images – e.g., the waist, and to estimate how much time participants spend fixating in these areas. One problem with this AOI approach is that across a range of stimuli, even though the ‘waist’ area may be defined according to consistent anatomical landmarks, it may well constitute different sized physical areas on the stimulus display, because some people are larger / differently proportioned than others. Uncontrolled, such inequality across stimuli can lead to sampling bias in the statistical analyses. Another problem is that by defining an AOI a priori, its spatial extent may (by chance) end up including some sub-regions where group A looks more than group B and other sub-regions where group A looks less than group B. Therefore, when averaged across the whole AOI, there may be no apparent differences between groups A and B.

To avoid both of these problems, in the current study, we used the same data driven approach to localisation as George et al. (2011) which is very similar to the analysis of neuroimaging data. In collaboration with Peter Hancock at Stirling University, we used a morphing procedure (Hancock, 2000) to define a reference stimulus image onto which all the stimuli, as well as the eye movement data, can be spatially co-registered. This means that while the co-ordinates for a feature like the left eye pupil may differ across individual stimuli, they will all be the same in the reference image, after co-registration. And the same is true for any eye fixations to the left eye of any individual stimuli. Because of the spatial co-registration, we can apply a much finer sampling grid (in our case each bin of the grid is a 20x20 pixel square) to the entire image in the reference space; in effect a 2D lattice of contiguous square AOIs. This cell size (20x20 pixels) represents a compromise between capturing as many fixation samples per cell as possible to optimize statistical power (which ideally requires large cells) versus retaining good anatomical resolution (which ideally requires small cells). Therefore, what might before have been a single, relatively large AOI in the waist area (if defined a priori), now becomes a subset of that sampling space, corresponding to all the squares in the waist area, as well as all their neighbours.

George et al. (2011) showed that the distribution of fixation durations in each cell of the sampling grid is rarely normal, but that the correlation between fixation duration and fixation count is very high (typically $r > 0.9$). Therefore, as in this previous work, we chose fixation counts per cell (also known as fixation density) as our outcome variable. We modelled differences in fixation counts between groups by applying generalized linear mixed models (GLMMs). To do this we used PROC GLIMMIX in SAS v9.3.

In each of the three statistical models comparing two groups [i.e., ANSD vs CN(ACC); ANSD vs CN(OVER); CN(OVER) vs CN(ACC)] we not only took account of the repeated measures factors – i.e. each subject contributed a number of fixations to the sampling grid (defined by row and column indices in the model) for each of the 110 images, but we also controlled for spatial co-variance by incorporating the spatial variability into the statistical models. We assumed the fixation counts to follow the Poisson distribution and consequently a log-link was used as a link function in the models for the outcome. The spatial variability was integrated into the models by specifying a Gaussian spatial correlation model for the model residuals. The GLIMMIX procedure was then used to assess where on the stimulus images there were significant differences in fixation density between the groups. Areas of significant difference are indicated by the white contours ($p < .05$) in Figure 7.1, and are based on the estimated marginal means derived from the model parameters. These predicted population margins are compared using tests for simple effects by partitioning the interaction effects, and are controlled for multiple comparisons. Finally, interpreting differences in fixation density / count is not necessarily straightforward. Therefore, to provide a more biologically interpretable sense of effect sizes in the areas where we found the most robust, statistically significant differences, we also report mean fixation durations (i.e. for the boundaries marked A, B, C, D & E as indicated by the arrows in the difference maps in Figure 7.1). To do this, we converted the total fixation density in the set of sampling cells contained within each highlighted statistical boundary into estimates of fixation duration per subject, separately for each group that contributed to the difference. We also marked with a black dot the location of the maximum difference within each of these highlighted boundaries.

7.3. Results

7.3.1. Univariate statistics

The inter-rater reliability of participants' responses to the psychometric questionnaires across the sample was high: Cronbach's alpha for BSQ, EAT, EDE-Q, RSE and BDI was: .96, .92, .96, .89 and .93 respectively.

The right hand columns of Table 7.1 show the output of pairwise comparisons of the group means for the psychometric and psychophysical measures, adjusted for multiple comparisons, using the permutation method in PROC MULTTEST (SAS v9.3). Table 7.1 shows that there are no statistically significant differences between the two control groups on any measure, except that, as intended, CN(OVER) and ANSD participants over-estimated their body size whereas CN(ACC) participants did not. Table 7.1 confirms that ANSD participants showed significantly elevated concerns about body shape and weight, reduced self-esteem and an increased tendency towards depression compared to both control groups. The mean BSQ score for the ANSD participants is above the critical threshold of 66 for eating disorder (Evans and Dolan, 1993), and the mean EDE-Q global score for this group is above the 90th percentile for young women in the UK (Mond et al., 2006) and for female undergraduates (Luce et al., 2008). Consistent with the requirement to control for individual BMI, as discussed in the Introduction to this chapter, the BMI of ANSD participants did not differ significantly from either control group. Finally, Table 7.1 confirms that there were no statistically significant differences in task sensitivity (DL) between the three participant groups. Overall, this pattern of performance on the psychometric tasks is consistent with a sample of female participants who have recovered / are recovering from anorexia nervosa and who

over-estimate their body-size, to be compared with two groups of healthy, non-eating disordered adult females, one of which over-estimates body size and the other of which does not.

7.3.2. Multivariate statistics

7.3.2.1. Behavioural responses in psychophysical tasks

Across all 60 participants, the correlation between PSE estimated from the eye movement recording sessions and the 2AFC task was high ($r=0.87$, $p<.001$). The mean over-estimation (i.e. PSE-BMI) computed from responses during the eye movement recordings were: 2.98, -0.46 & 1.41 for ANSD, CN(ACC) and CN(OVER) participants respectively. A one way ANOVA with participant group as a factor was statistically significant ($F_{2,57} = 9.65$, $p<.001$). We found statistically significant post-hoc comparisons between ANSD & CN(ACC) ($t=4.39$, $p<.001$) and CN(ACC) & CN(OVER) ($t=-2.25$, $p<.05$), but not between ANSD & CN(OVER) ($t=1.88$, $p>.05$). Therefore, despite the fact that the estimates of PSE obtained from the eye movement recording sessions are much coarser than those from the 2AFC task, they are nevertheless consistent with each other and confirm that participants performed during the eye movement recordings according to the groups to which they had been they assigned.

The distributions of participants' response times during the eye movement recording sessions were clipped at the mean ± 2 SD separately for each participant. Mean response times for ANSD, CN(ACC) and CN(OVER) participants were: 2505.6ms, 1917.4ms and 2354.1ms respectively. A one way ANOVA with participant group as a factor was statistically significant ($F_{2,57} = 7.38$, $p<.001$). Post-hoc Bonferroni pairwise comparisons between ANSD & CN(ACC) and

CN(OVER) & CN(ACC) were both statistically significant ($t=3.71$, $p<.005$ and $t2.72$, $p<.05$ respectively). However, the comparison between ANSD & CN(OVER) was not ($t=0.94$, $p>.1$). These results suggest that accurate body size estimation took approximately 1 to 2 fixations less than inaccurate estimation, and critically, that participants appeared to make their decisions after ~2 to 2.5 seconds. Consistent with George et al. (2011), this therefore justified restricting the eye movement analysis to the first 2000ms following stimulus onset.

7.3.2.2. Comparisons of fixation density between groups

The analysis of the eye movement data excluded the first 400ms of each trial and the gaze patterns beyond the time window of 2000ms. Figure 7.1 shows the fixation patterns for each individual group: ANSD; CN(ACC) and CN(OVER) as well as the differences between them. The individual maps show clearly that all participants spend more time looking in the abdominal region than anywhere else. However, the maps also show clearly that the tendency to look outside this region, particularly towards the head and chest, increased systematically from CN(ACC) through CN(OVER) to ANSD. These differences, which are apparent on inspection, were confirmed by the statistical group comparisons shown in Figure 7.1 overleaf.

The estimates of r-square for the three models comparing: ANSD vs CN(ACC); ANSD vs CN(OVER) and CN(OVER) vs CN(ACC) were 0.62, 0.55 and 0.59 respectively. The white boundaries, indicating regions of statistically significant difference in Figure 7.1, show that ANSD participants looked significantly more at the face than did the CN(ACC) group (Figure 7.1, boundary A: ANSD = 180.7(76.5) ms/subject; CN(ACC) = 48.5(19.1) ms/subject). By contrast, ANSD participants looked in the central abdominal region significantly less than the CN(ACC) group

(Figure 7.1, boundary B: ANSD = 186.2(45.5) ms/subject; CN(ACC) = 381.3(82.3) ms/subject).

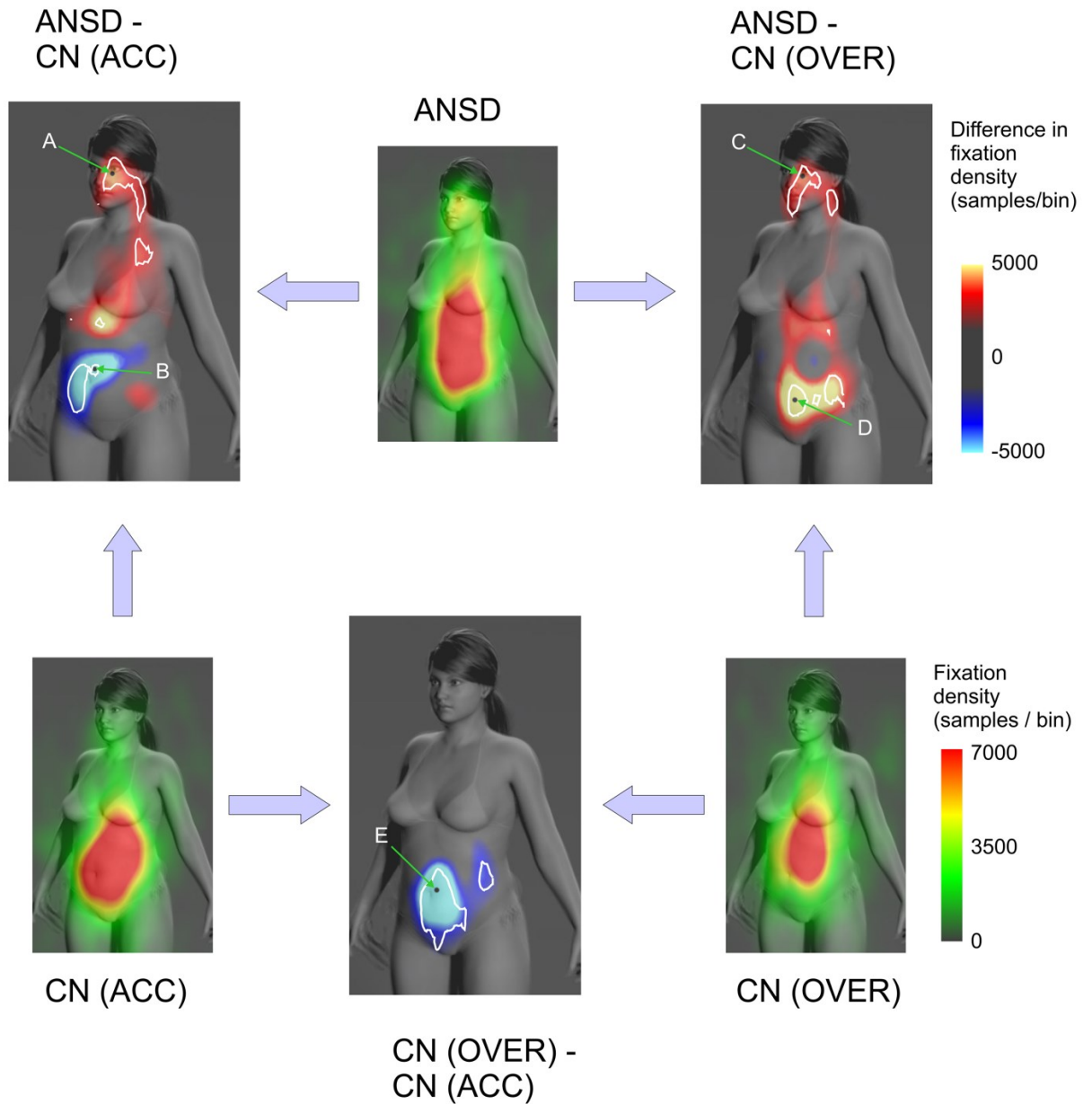


Figure 7.1: Maps of the relationship between psychophysical performance in body size estimation and where observers look on the body. The colour scales of the heat maps are expressed in terms of fixation counts or differences in fixation counts per sample bin. The heat maps with the green/yellow/red colour scales illustrate the pattern of looking for each of the three groups in isolation. The difference maps with

the cyan/blue/red/yellow colour scales illustrate the differences between pairs of groups of participants, indicated by the arrows. Blue/cyan colours indicate where accurate observers looked more frequently than over estimators. Red/yellow colours indicate where over estimators looked more frequently than accurate observers. White lines indicate the regions within which the comparisons between groups were statistically significant at $p < 0.05$ (corrected for multiple comparisons), as defined by the spatial statistical modelling with PROC GLIMMIX (SAS v9.3). The labelled arrows indicate those statistical boundaries for which we report mean fixation duration per subject for each of the groups contributing to a particular comparison (see text for details). The black dots indicate the location of the maximum difference in fixation density for that labelled statistical boundaries.

The ANSD group looked at the face significantly more than the CN(OVER) group (Figure 7.1, boundary C: ANSD = 218.7(77.4) ms/subject; CN(OVER) = 62.2(16.1) ms/subject). In addition, the ANSD group looked in the abdominal region significantly more than the CN(OVER) group (Figure 7.1, boundary D: ANSD = 459.6(160.5) ms/subject; CN(OVER) = 180.3(46.4) ms/subject). Finally, the CN(OVER) group looked significantly less in the central abdominal region than the CN(ACC) group (Figure 7.1, boundary E: CN(OVER) = 227.3(49.7) ms/subject; CN(ACC) = 588.2(99.6) ms/subject).

7.3.2.3. Fixation density as a function of stimulus BMI

We wanted to know whether the three different groups of participants adopted different fixation patterns depending on the BMI of the stimulus image being viewed. To address this question, we divided the 11 stimulus images into three

sub-groups: low, middle and high BMI, each of which comprised three images per group, as is shown in Table 7.2 below.

Table 7.2: Stimulus images contributing to three BMI sub-groups

Image	BMI	Mean BMI (from 3 images)	BMI sub- group
1 2 3	12.5 15.5 18.5	15.5	Low
4 5 6	21.5 24.5 27.5	24.5	Med
7 8 9 10 11	30.5 33.5 36.5 39.5 42.5	(discarded) 36.5 (discarded)	High

We discarded two of the images to make the high BMI sub-group so that sampling density over the 2000ms of fixations was equated between sub-groups. Figure 7.2 overleaf, shows plots of fixation density for the three groups ANSD, CN(ACC) and CN(OVER) in columns 2, 3 & 4, split according to whether the data came from low, middle or high BMI images (i.e. rows 1, 2 & 3). Column one also shows one set of difference plots for the comparison ANSD – CN(ACC). The over-riding impression given by these data is that image BMI had no obvious influence on fixation density, and that the plots are markedly similar across the three groups of participants. We acknowledge that a formal statistical analysis of all the possible differences might well reveal small regions of statistically significant difference between some pairings, but these would be likely to be difficult to interpret and, given the large

number of possible comparisons, likely to represent false positive , Type I errors. More than anything, such differences might detract from the main impression of similarity.

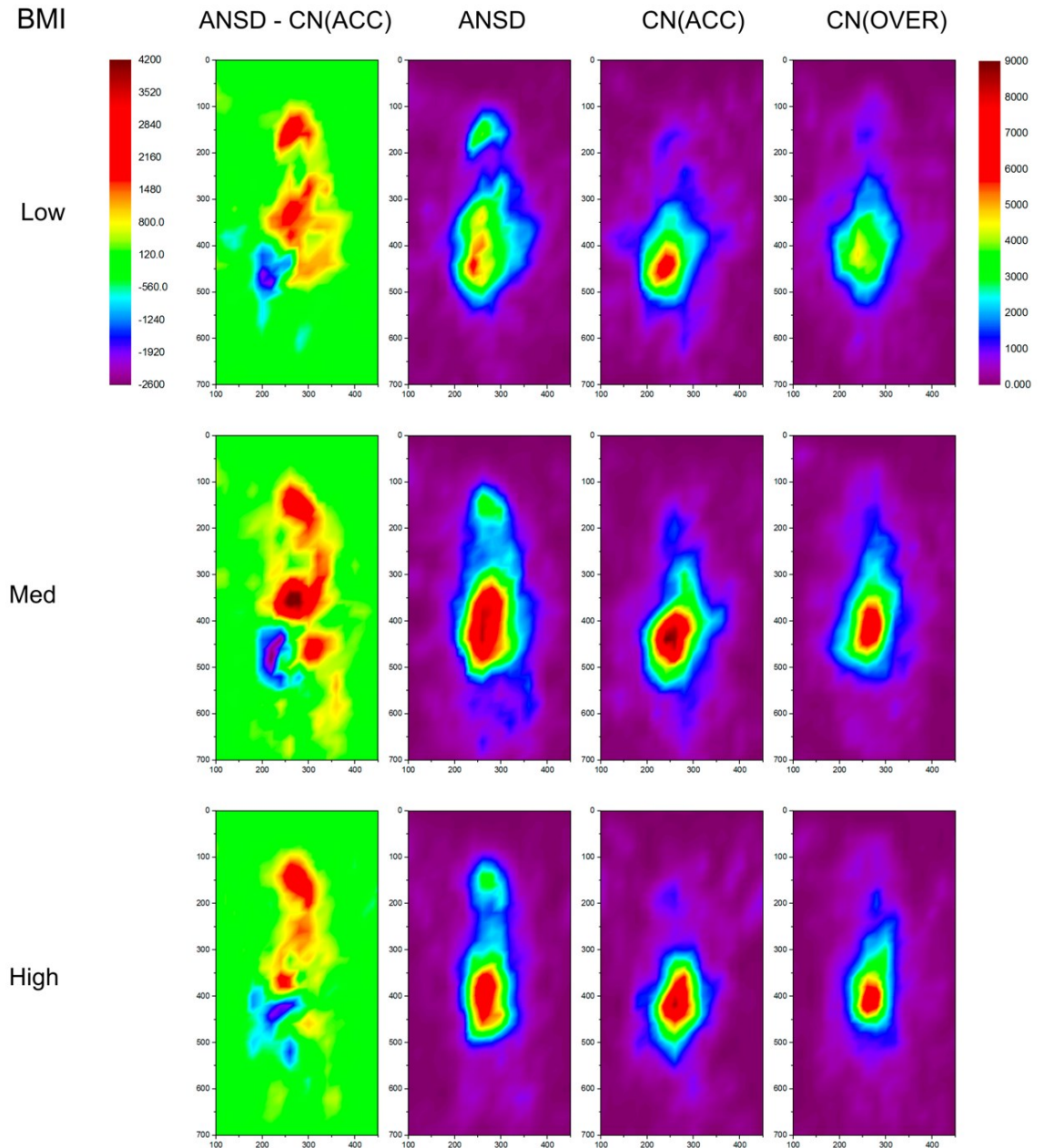


Figure 7.2: Fixation density as a function of stimulus image BMI grouping.

7.4. Discussion

In this eye movement study, the results suggest that, irrespective of AN diagnosis, accurate body size estimators looked more in the central abdominal region than over-estimators did. Therefore, individuals who are capable of making an accurate estimation of body size spend more time looking in regions of the body that are most informative about body size – i.e. the waist (see, e.g., Cornelissen et al., 2009b; Wells et al., 2007; Wells, Cole, Bruner & Treleaven, 2008). The fact that this is true for both the ANSD vs CN(ACC) and CN(OVER) vs CN(ACC) comparisons, suggests that this difference in the relationship between perceptual performance and gaze cannot be accounted for by psychological factors – at least with regard to the abdominal region. In other words, it does not appear to be a feature that is specific to anorexia nervosa; anyone who can estimate body size accurately tends to focus their attention on the central abdomen. By comparison, there is another effect which does appear to be specific to those with AN, namely the tendency to look more in the chest and facial regions. This is revealed as a significant difference between ANSD v CN(ACC) as well as ANSD v CN(OVER) in Figure 7.1, but not CN(ACC) v CN(OVER).

7.4.1. The role of the face

A number of studies have suggested that changes in facial shape are correlated with overall body mass and that it is possible to estimate overall body mass from the face alone (Coetze, Jingyang, Perrett & Stephen, 2010; Coetze, Perrett & Stephen, 2009). However, the relationship between facial shape and the BMI of the individual is weaker than is found for the equivalent torso shape change, and judgements of overall body mass based on facial cues are also less precise than

judgements based on the rest of the body (Coetze et al., 2010). This suggests that using the face to judge overall body fat is a suboptimal strategy for estimating body mass and its fixation in preference to the torso may lead to an inaccurate judgement. An alternative explanation is that the eye-movements towards the face represent a bias toward sampling of socially important information (Tapajóz, de Sampaio, Soneira, Aulicino, Harris, 2015), although there is some evidence for women with AN actually avoiding faces (Watson, Werling, Zucker & Platt, 2010). However, although the faces in this study vary in adiposity, no other features (such as expression, gaze direction or identity) change and the participants are familiar with the images and are aware of this. So it seems unlikely that this is an explanation for why our participants with ANSD looked more at the faces.

7.4.2. Potential confounds in group comparisons

In this study we sought to eliminate possible confounds between our observer groups which might obscure the basis of the body size estimation. The lack of a difference in the BMI of the women in the three groups is important. As Experiments 1 and 2 showed, in Chapter 3, the BMI of the body being judged affects the accuracy of estimation due to contraction bias and Weber's law. In previous studies comparing the results of controls and women with AN, the two sets of participants usually have very different BMIs, and as they are estimating the size of their own bodies, the two groups are likely to be estimating the size of significantly different bodies. Therefore, it is possible that these normal perceptual biases may have been responsible, at least in part, for any group differences observed. In this study there were no differences in the BMI of the bodies being judged, nor, on average, between observer groups, and so this source of error in estimation was removed.

The neural changes, cognitive impairments and low level motor impairments in eye-movements that have been found in low BMI women with AN as a result of malnutrition largely disappear with refeeding (Joos, Klöppel, Hartmann, Glauche, Tüscher et al., 2010; King, Geisler, Ritschel, Boehm, Seidel et al., 2014; Pallanti, Quercioli, Zaccara, Ramacciotti & Arnetoli, 1998; Phillipou, Rossell, Castle, Gurvich & Abel, 2014). Therefore, because the BMI of the ANSD group was comparable to that of the two control groups, their fixation patterns are unlikely to be due to cognitive impairment or their inability to move their gaze across the image as they would wish.

Some previous studies of attractiveness judgements have reported that women with AN look less at the breast region than control observers, a result interpreted as reflecting an avoidance of areas they dislike or have concerns about (Hewig et al., 2008; von Wietersheim et al., 2012). This pattern of fixations differs from the results reported here. This could be because one of these studies has focused on the perception of attractiveness rather than adiposity (von Wietersheim et al., 2012), and there is evidence of differences in the pattern of fixations for these two judgements (Cornelissen et al., 2009a; George et al., 2011). However, George et al. (2011) also found that when making attractiveness judgements, women with AN looked more at the upper torso than controls. Another possible reason for this discrepancy is that Hewig et al. (2008) and von Wietersheim et al. (2012) recorded eye-movements during a “free-viewing” of bodies. As their participants were not asked to make a specific judgement during the eye-tracking, it is not possible to say with certainty what judgement, if any, their participants were making. This might result in differences relative to the fixations made during a specific judgement such as rating bodies for attractiveness or size. However, avoidant behaviour could

indeed be an explanation of the more distributed pattern of eye-movements made by women with AN or ANSD. Regions of potential sensitivity, such as the stomach, may be avoided by women with ANSD or AN, resulting in a pattern of eye-movements spread up along the torso as reported here and by George et al. (2011).

7.4.3. Perceptual versus cognitive factors

The design of this study reveals eye movement patterns in the lower abdominal region that are associated with over-estimation of body size. This association may be causal, but, given the experimental design, we cannot claim that it definitely is or is not. Nevertheless, we can reasonably claim that through our between group comparisons we can eliminate attitudinal factors / psychological concerns about body shape as a source of the over-estimation, since the over-estimating controls do not have these concerns.

It is possible that the patterns of eye-movements made by the CON(OVER) and ANSD groups arise from different causes. The pattern of eye-movements made by ANSD participants may arise from attentional biases or the pattern of fixations may be due to a specialisation for discriminating between very low BMI bodies (such as detecting the bony landmarks). This idea was first suggested in the Discussion of Experiment 3, Chapter 4 in the form of an expertise effect. Consistent with this idea, we found that ANSD participants tended to look significantly more at faces than either of the two control groups who may simply represent two ends of a normal distribution in size judgement ability. The fixations on the upper chest and face may not be good predictors of overall body fat across the whole BMI range, but the prominence of the underlying bone structure at very low BMI values may provide cues to how thin the body has become.

7.4.4. Limitations

One limitation of this study is that the bodies used in this experiment are artificial using a simulation of body mass change based on the average pattern of fat deposition rather than real bodies varying in BMI. The advantage of the CGI bodies is that only body mass is changing across the set of stimuli, while skeletal proportions, skin texture and identity are held constant. However, we acknowledge that responding to ‘virtual’ stimuli may engender different patterns of response, compared to stimuli drawn from the real world. Nonetheless the results reported here are consistent with the pattern of fixations made when recording from observers estimating the body size of photographs of real women (Cornelissen et al., 2009a; George et al., 2011). Another potential limitation is the inclusion of the face and head in the stimuli, so they were not just body stimuli. However, we would argue that the inclusion of the head makes it a more ecologically valid stimulus (you seldom see bodies without their heads) and features such as the identity, expression, gaze direction and hairstyle are constant across all the images in the stimulus set so the only differences between the faces and heads are the change in adiposity. A final limitation is that the BMI of the women in this study is not below 18.5, which is one of the DSM-5 criteria for AN. It might be argued that to properly measure the pattern of eye-movements in an AN population, we should have used a group of women who fully conformed to the DSM-5 criteria. However, to avoid the perceptual factors linked to observer BMI that confound such a comparison, it would have been necessary to recruit over-estimating and accurate control groups of the same low BMI. Such individuals are unlikely to have such a low BMI without a medical cause which in turn would introduce new confounding factors into the comparison.

7.4.5. Treatment implications

Our results suggest a significant association of fixation patterns with the accuracy of body size judgements, which is unrelated to the psychological or physical state of the observer. If there is a causal link, this suggests that a perceptual training regime to improve the accuracy of body estimation could be an effective adjunct to conventional treatment with cognitive behavioural therapy. One potential way of treating this problem could use a training programme incorporating gaze contingent eye-tracking (i.e. incorporating a feedback loop from the eye-tracking to the positioning of the stimulus on the computer monitor) to shift the fixation pattern in women with AN towards the pattern seen in accurate control observers to improve body size estimation accuracy. Given the encouraging outcomes from the training studies in Experiments 5 and 6, Chapter 6, a future study might test whether the efficacy of training could be further improved by adding an eye-movement contingent component to the perceptual training task.

Chapter 8

Summary and General Discussion

8.1. Overview

This chapter summarises and discusses the main findings from the experiments presented in this thesis. Theoretical implications of the findings are discussed and future directions are proposed.

8.2. Research aims

The weight of evidence presented in the Introduction suggested that women with AN do indeed over-estimate their body-size, on average, when compared to healthy controls. However, there is less consensus about the relative contributions of attitudinal and perceptual factors to body image distortion. Moreover, questions remain about what are the mechanisms that drive the perceptual contribution to body image distortion in particular. Are perceptual factors really acting as proxies for attitudinal factors? Are perceptual factors different from normal visual psychophysical biases? What role does BMI play? In addition, it was clear that methodological differences between studies may have contributed to this confusion, whether they arose as a result of sub-optimal stimulus design or failure to apply rigorous psychophysical methodology. Therefore, the primary aim of the research reported in this thesis was to try to obtain a more coherent account of body image distortion in AN by simultaneously measuring: (i) the perceptual component using the most ecologically valid stimuli we could build together with robust psychophysical methods, (ii) the attitudinal contribution of body size estimation using a battery of well validated, normed psychometric tools, (iii) taking account of

the BMI of the observer. Having obtained such a description, we then tested a novel approach to the treatment of body image distortion.

8.3. Summary of research findings

8.3.1. Chapter 3

In Chapter 3, two visual psychophysical studies were presented to confirm whether two normal visual biases in magnitude estimation, contraction bias and Weber's law, apply when observers are judging the body size of others, as we would predict they should. In Experiment 1, participants were shown a photograph of a woman on each of 120 trials, and they were asked to move an on-screen slider to estimate the body weight of each woman. A multivariate analysis of the relationship between these estimates of body weight, and the actual weight of the women showed clear evidence of contraction bias in the estimation of human body size (Poulton, 1989). In Experiment 2, we used the method of constant stimuli with pairs of images presented on each trial, to measure the just noticeable difference in BMI as a function of stimulus BMI. For CGI models of human females, as well as photographs of real women, we found evidence consistent with Weber's law (Gescheider, 1997).

8.3.2. Chapter 4

In Chapter 4, data were presented from an experiment in which healthy control participants and women with ANSD used the method of constant stimuli to estimate their body size. Both the degree of over-/under-estimation of body size, as well as their sensitivity to differences in body size were statistically modelled on the basis of group assignment, chronological age of the participants and their

psychometric performance in tasks assessing concern about body shape and weight, tendency to depression and self-esteem. The key findings of Chapter 4 were:

- Control participants showed clear evidence of contraction bias in their body-size estimates, and Weber's Law behaviour in their task sensitivity.
- Women with ANSD who had the lowest BMI were very accurate at the size estimation task, and extremely sensitive to BMI difference. However, as this group's BMI increased, so their sensitivity reduced dramatically and they increasingly over-estimated body size.
- Both participant groups showed an equivalent effect of over-estimation which depended on their psychological concerns, and was independent of BMI.

8.3.3. Chapter 5

In Chapter 5 we carried out a very similar, albeit smaller scale, study to that reported in Chapter 4. The main differences were that participants were asked to carry out the method of limits, and the stimuli they were judging represented avatars of themselves, which were built from individual 3D body shape scans. The key findings of Chapter 4 were:

- Neither healthy control participants, nor women who had largely recovered from AN and who were no longer being treated, showed any evidence of contraction bias in their body-size estimates
- Women with AN who were still being treated showed a pattern of responses similar to that in Chapter 4; i.e. accurate estimation at the lowest BMI, with increasing over-estimation of body size as BMI increased.
- Estimates of ideal body size/shape showed an almost uniform response from healthy controls in which they all desired lighter bodies. The magnitude of

this reduction was greatest for individuals with the highest BMIs. Both groups of women with ANSD desired ideal bodies that were ~1.3 BMI units lighter than the body they owned.

8.3.4. Chapter 6

Chapter 6 presented two experiments in which a perceptual training paradigm was used to shift observers' categorical boundaries for thin/fat body shape towards a higher BMI. This was successfully achieved in (i) undergraduate observers with high body shape concerns and (ii) a group of women with AN who were receiving treatment as outpatients. In both experiments, we observed significant reductions in body shape and weight concerns, estimated by the EDE-Q questionnaire, and these effects persisted for up to a month post-training in the women with AN.

8.3.5. Chapter 7

Chapter 7 presented the final experiment in which we recorded the eye movements of three groups of participants while they were carrying out the method of constant stimuli to estimate their own body size: (i) women with ANSD, who were known to over-estimate body size, and who also had elevated body shape concerns; (ii) healthy controls, with no body shape concerns, who assessed their body size accurately; (iii) healthy controls, with no body shape concerns, who over-estimated their own body size. The key findings of Chapter 7 were:

- Accurate body size estimators tended to look more in the waist region, and this was independent of clinical diagnosis.

- There is a pattern of looking at images of bodies, particularly viewing the upper parts of the torso and face, which is specific to participants with ANSD but which is independent of accuracy in body size estimation.

8.4. General discussion

Overall, the findings from Chapters 3, 4 and 5 support a model of body image distortion which comprises independent contributions from: (i) the perceptual domain, (ii) the attitudinal domain, (iii) the physical domain in the form of an individual's BMI. Arguably, one of the most important contributions from this thesis is the confirmation of the contribution of BMI to this model. Moreover, how these three factors interact with each other in order to predict whether an individual will over- or under-estimate their body weight depends on whether an individual has a history of eating disorders or not. For both women with ANSD, as well as healthy controls, attitudinal factors indexed by the psychometric variables we measured in this thesis determine one of these dimensions – i.e. body size over-estimation increases linearly as a function of increasing concern about body shape, weight, tendency to depression and low self-esteem. For healthy controls, an individual's BMI determines the perceptual contribution to body size estimation in a way that can be explained by a combination of contraction bias and Weber's Law behaviour – both of which are perfectly normal attributes for perceptual magnitude estimation. Interestingly, we found that if a 'model of another person' was replaced with a realistic avatar of the participant, as the stimulus in the task to estimate body size, then contraction bias at least seems to disappear. So, under these test circumstances,

for healthy controls at least, the perceptual biases may be removed or substantially reduced.

The effect of BMI on the perceptual component of body size estimation in women with ANSD is rather different to that of healthy controls, and, arguably it is another of the most important results in this thesis. Women with ANSD who had the lowest BMI estimated their body size very accurately. However, as this group's BMI increased, so their sensitivity reduced dramatically and they increasingly over-estimated body size. We suggest that this result could be explained by an expertise effect, and that it has important implications clinically.

8.4.1. Clinical implications

Women with ANSD whose BMI was less than ~ 17.5 , were either very accurate at body size estimation, or if anything tended to slightly under-estimate. Therefore, with respect to the maintenance of an eating disorder, this finding suggests that body size over-estimation may have very little effect at low BMI levels. Since such individuals are so sensitive to changes in body shape and size, it is very likely that they will easily detect changes in their weight. In turn, this may then stimulate a renewal of weight reduction behaviours. However, we found that with increasing BMI, sensitivity to body size change reduces dramatically in women with ANSD, and much faster than is the case in healthy controls. We suggest therefore, that this may have important implications for potential relapse in recovering patients. Perhaps owing to the expertise effect we suggested in Chapter 4, such women with ANSD will see themselves as larger than they actually are, and a substantial part of this over-estimation will be based on perceptual factors not easily treated through talking therapies such as cognitive behavioural therapy (CBT). Given the core nature

of body size concerns for AN (Cash & Deagle, 1997; Fairburn et al., 2003; Fairburn & Harrison, 2003), this represents a potentially important factor in any subsequent relapse and may contribute to the relatively high relapse rate (Channon & De Silva, 1985; Slade & Russell, 1973). However, this is not to say that CBT cannot play a role in reducing body size over-estimation. For a given BMI, our results suggest that the degree of over-estimation by women with ANSD is modulated by their body size and shape concerns. So talking therapies such as CBT can potentially reduce the over-estimation of body size by targeting these concerns. However, to improve perceptual judgements, the perceptual training technique that we investigated in Chapter 6 may prove to be helpful as an adjunct to standard therapy. Clearly, an early Phase 2 trial of this approach would be required to test this hypothesis. Should this prove successful, given the results from Chapter 7, then further research could test whether adding in an eye fixation training component would prove even more effective.

8.4.2. Limitations

There are probably two major limitations to the studies reported in this thesis. As mentioned at the end of Chapter 4, the first limitation is that all of the studies reported in this thesis, with the exception of the intervention study in Chapter 6, are correlational in design. Therefore, it is not possible to make definitive causal claims of the data, particularly with respect to any of the mechanisms proposed to explain distorted body image in ANSD. Future intervention or longitudinal studies will therefore be needed. With regard to the intervention study (Chapter 6), it is also questionable whether the reduction in psychological concerns about body image (indexed by EDE-Q scores) as a result of perceptual training – however persistent – will be enough to persuade clinicians to include this technique (or something like it)

in their clinical practice. However, were we to demonstrate in a future study that perceptual training led not only to a reduction in psychological concerns, but also to a reduction in relapse rates, perhaps even as modest as 10-15%, then this may be sufficient to have an impact on clinical practice.

The second major limitation to the studies reported here is in regard to the choice of stimuli that we have used in all our studies. In the Introduction we claimed that many of the stimuli that have been used in the past to measure body size have lacked ecological validity, and have even introduced image distortions that do not occur in reality (e.g., in the Video Distortion Technique). To our knowledge, there seem to be three solutions to these problems. One is to use images which accurately capture the central statistical tendencies for how body shape changes as a function of BMI.

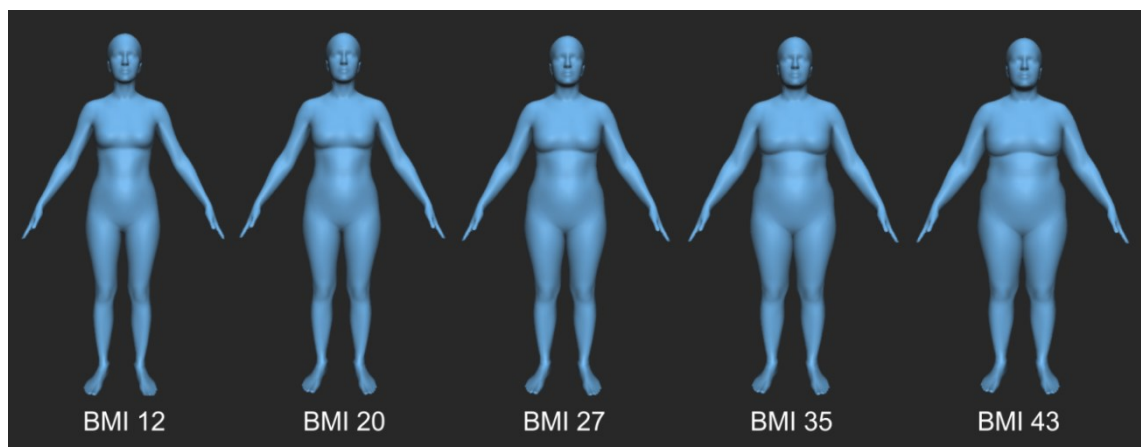


Figure 8.1: Representations of body shape change in a female, aged 25, height 1.6m, as a function of BM, courtesy of Perceiving Systems, MPI, Tübingen. These representations are derived from a statistical model based on a large number of 3D body scans (cf. Hasler et al. 2009).

While these may be accurate representations statistically, such images, as illustrated in Figure 8.1 above, inevitably incorporate a great deal of smoothing, and arguably may make it more difficult for participants to relate the image they are being shown to their mental representation of their own body image. By analogy, related difficulties have been shown with respect to judgements made about the attractiveness of symmetrical versus asymmetrical faces; observers' preferences depend heavily on the way that skin texture is processed (Perrett, Burt, Penton-Voak, Lee, Rowland & Edwards, 1999)

The second option, which we chose for our studies, is to use images that are good representations of body shape changes in one "model" individual, that are photorealistic, and where those changes can be calibrated for BMI. While this solution ought to have greater ecological validity – most of us have been to beaches and swimming pools where we see many examples of other peoples' bodies - this strategy introduces fixed biases to the task which may be specific to the particular model in question. The fact that we could not find a statistical difference as a function of CGI model (i.e. Victoria 5 versus Victoria 6) or body shape morphs (i.e. Genesis versus Genesis 2) in Experiment 2a, in Chapter 3, provides some reassurance that this problem may not be too concerning. Nevertheless, we cannot avoid it.

A variant of the second option is to use photographs of many different individuals as stimuli, in the hope that the signal we want to measure – i.e. body shape change related to changing adiposity – can be pulled out of the additional noise produced by the fact that skeletal shape changes, height, muscularity and many other features vary across individuals. To do this reliably would therefore require many more participant responses, from many more stimulus presentations, and

possibly make undue demands of participants' tolerance. Evidence that this is likely to be the case comes from the fact that not only were the estimates of just noticeable difference (JND) in body size increased for photos, compared to CGI stimuli, in Experiment 2b, Chapter 3, but the steepness of the function relating JND to BMI was steeper than would normally be consistent with Weber's Law.

The third option, which we attempted in Chapter 5, is to use stimuli which are specific to each individual participant, because they are based on a 3D body scan of that person. In principle, this reduces the problem that some participants have body shapes that are inherently closer to a standard model than other participants. Therefore, this strategy 'normalizes' whatever are the cognitive transformations needed to map between the externally presented stimulus and an individual's mental representation of their body shape. However, it does not avoid the problem that we still had to use the same set of shape morphs to represent changing adiposity across all models.

8.4.3. Future research: the importance of body ownership

Intuitively, when we look at ourselves in the bathroom mirror, it is clear that we know that we own and inhabit the body whose reflection we see. However, in the laboratory, participants usually make judgements about stimuli presented on a monitor from a third person point-of-view (POV-3). This was certainly the case in Chapters 3, 4, 6 and 7 in this thesis. However, recent research by Preston and Ehrsson (2014) has made it very clear that POV has a critical effect in judgements of body image. The third person perspective (POV-3) means participants make a judgement about their own body image in relation to another person in the image "over there" on the screen. The first person perspective (POV-1) means that

participants believe that the stimulus they are looking at “is me, in my body here”. To achieve this transfer of POV from third to first person perspective, Preston and Ehrsson arranged that a wider or narrower video projection of a person’s body was projected into a virtual reality (VR) headset as if the camera was in the same location as the participant’s eye. Therefore, when participants looked down towards their feet, they saw fatter or thinner versions of themselves. The illusion was completed by combining the VR presentation with an animation in which the participant could see themselves being touched by the experimenter, and this sight and proprioceptive experience was either synchronous or asynchronous with the video animation. Previous research by Ehrsson (2007) has demonstrated that the synchronous experience induces a more compelling transfer from third to first person POV. Critically, Preston and Ehrson (2014) found, for example, that “inhabiting” a slimmer body resulted in participants judging *their actual* body as slimmer, and as a result they gave higher ratings of body satisfaction than they did before the POV manipulation. A similar, but more thorough investigation was carried out by Piryankova, Wong, Linkenauger, Stinson, Longo et al. (2014). These authors found that the sense of agency – or body ownership – did not critically depend on synchronous / asynchronous touching, probably because they were using a VR avatar which moved in real time and the integration of proprioceptive and somatosensory feedback from the participant’s physical body together with the cues provided from the head tracking and the visual stimuli may have sufficed to induce ‘presence’. In addition, these authors further quantified body ownership by introducing an affordance measure – judging whether two poles were just close enough to walk through (indirect measure of body size) and body size (direct measure of body size) estimations, carried out from a third person POV. Their results clearly showed that

participants perceived a change in their experienced body dimensions consistent with the thinner or fatter virtual body they were induced to inhabit.

Together, this research suggests that future investigations of what it means to have distorted body image in AN will require not only that experimental stimuli are as ecologically valid as possible – hence the probable need to create individual avatars of participants as in Chapter 5 - but that the estimations of body size need to be made from both third (POV-3) and first (POV-1) person perspectives, and compared to affordance estimates, in order that we establish whether POV plays a necessary role in body image distortion or not.

8.5. Conclusions

Despite some methodological limitations, the results from the experiments in this thesis have shed new light on what it means to have distorted body image in anorexia nervosa. In particular, we have revealed the central role that an individual's current BMI plays in the predicting relationship between body size over-estimation, perceptual and attitudinal factors. Moreover, we present evidence from an intervention study to suggest new therapeutic options to help reduce body image distortion and its associated psychological concerns.

References

- Adams, S., Penton-Voak., I.S., Harmer, C.J., Holmes, E.A., & Munafo, M.R. (2013). Effects of emotion recognition training on mood among individuals with high levels of depressive symptoms: study protocol for a randomized controlled trial. *Trials*, 14, 161.
- Aeberli, I., Gut-Knabenhans, M., Kusche-Ammann, R.S., Molinari, L., & Zimmermann, M.B. (2011). Waist circumference and waist-to-height ratio percentiles in a nationally representative sample of 6-13 year old children in Switzerland. *Swiss Medical Weekly*, 141:w13227. doi: 10.4414/smw.2011.13227
- Agras, W. S., Bryson, S., Hammer, L. D., & Kraemer, H. C. (2007). Childhood risk factors for thin body preoccupation and social pressure to be thin. *Journal of the American Academy of Child & Adolescent Psychiatry*, 46(2), 171-178.
- Allebeck, P., Hallberg, D., & Espmark, S. (1976). Body image-An apparatus for measuring disturbances in estimation of size and shape. *Journal of Psychosomatic Research*, 20, 583-589.

Alleva, J.M., Sheeran, P., Webb, T.L., Martijn, C., & Miles, E. (2015). A Meta-Analytic Review of Stand-Alone Interventions to Improve Body Image. *PLoS ONE*, 10, 9: e0139177.

American Psychiatric Association (2006). *Treatment of Patients with Eating Disorders*. 3. American Psychiatric Association.

Anderson, L. A., Eyler, A. A., Galuska, D. A., Brown, D. R., & Brownson, R. C. (2002). Relationship of satisfaction with body size and trying to lose weight in a national survey of overweight and obese women aged 40 and older, United States. *Preventive medicine*, 35(4), 390-396.

Anderson, D.A., Shapiro, J.R., Lundgren, J.D., Spataro, L.E., & Frye, C.A. (2002). Self-reported dietary restraint is associated with elevated levels of salivary cortisol. *Appetite*, 38, 1, 13-17.

Askevold, F. (1975). Measuring body image: Preliminary report on a new method. *Psychotherapy and Psychosomatics*, 26, 71–77.

Augustine, J.R. (1996). Circuitry and functional aspects of the insular lobe in primates including humans. *Brain Res Brain Res Rev*, 22, 229-44.

Bär, K-J, de la Cruz, F., Berger, S., Schultz, C.C., & Wagner, G. (2015). Structural and functional differences in the cingulate cortex relate to disease severity in anorexia nervosa. *Journal of Psychiatry and Neuroscience*, 40, 4, 269-279.

Bardone-Cone, A. M., Harney, M. B., Maldonado, C. R., Lawson, M. A., Robinson, D. P., Smith, R., & Tosh, A. (2010). Defining Recovery from an Eating Disorder: Conceptualization, Validation, and Examination of Psychosocial Functioning and Psychiatric Comorbidity. *Behaviour Research and Therapy*, 48(3), 194–202.

Bardone-Cone, A. M., Wonderlich, S. A., Frost, R. O., Bulik, C. M., Mitchell, J. E., Uppala, S., & Simonich, H. (2007). Perfectionism and eating disorders: Current status and future directions. *Clinical Psychology Review*, 27(3), 384-405.

Bateson, M., Tovée, M. J., George, H. R., Gouws, A., & Cornelissen, P. L. (2014). Humans are not fooled by size illusions in attractiveness judgements. *Evolution and Human Behaviour*, 35(2), 133-139.

Beck, A. T., Ward, C. H., Mendelson, M., Mock, J., & Erbaugh, J. (1961). An inventory for measuring depression. *Archives of General Psychiatry*, 4, 561–571.

- Becker, A. E., Eddy, K. T., & Perloe, A. (2009). Clarifying criteria for cognitive signs and symptoms for eating disorders in DSM- V. *International Journal of Eating Disorders*, 42(7), 611-619.
- Bell, C., Kirkpatrick, S. W., & Rinn, R. C. (1986). Body image of anorexic, obese, and normal females. *Journal of Clinical Psychology*, 42, 431-439.
- Ben-Tovim, D. I., & Walker, M. K. (1991). Women's body attitudes: A review of measurement techniques. *International Journal of Eating Disorders*, 10, 155–167.
- Bergh, C., Brodin, U., Lindberg, G., & Södersten, P. (2002). Randomized controlled trial of a treatment for anorexia and bulimia nervosa. *Proceedings of the National Academy of Sciences*, 99(14), 9486-9491.
- Berkman, N.D., Lohr, K.N., & Bulik, C.M. (2007). Outcomes of eating disorders: a systematic review of the literature. *International Journal of Eating Disorders*, 40, 293-309.
- Bliss, E. L., & Branch, C. H. H. (1960). *Anorexia nervosa: Its history, psychology, and biology*. Hoeber [New York].

- Boraska, V., Franklin, C. S., Floyd, J. A., Thornton, L. M., Huckins, L. M., Southam, L., ..., & Lewis, C. M. (2014). A genome-wide association study of anorexia nervosa. *Molecular psychiatry*, 19(10), 1085-1094.
- Boyadjieva, S., & Steinhausen, H. C. (1996). The Eating Attitudes Test and the Eating Disorders Inventory in four Bulgarian clinical and nonclinical samples. *International Journal of Eating Disorders*, 19(1), 93-98.
- Brown, T. A., Cash, T. F., & Mikulka, P. J. (1990). Attitudinal body-image assessment: Factor analysis of the Body-Self Relations Questionnaire. *Journal of Personality Assessment*, 55, 135–144.
- Bruch, H. (1962). Perceptual and conceptual disturbances in anorexia nervosa. *Psychosomatic Medicine*, 24, 187–194.
- Bulik, C.M., Berkman, N.D., Brownley, K.A., Sedway, J.A., & Lohr, K.N. (2007). Anorexia nervosa treatment: A systematic review of randomized controlled trials. *International Journal of Eating Disorders*, 40, 4, 310-320.
- Bulik, C. M., Sullivan, P. F., Tozzi, F., Furberg, H., Lichtenstein, P., & Pedersen, N. L. (2006). Prevalence, heritability, and prospective risk factors for anorexia nervosa. *Archives of general psychiatry*, 63(3), 305-312.

- Bulik, C. M., Thornton, L. M., Root, T. L., Pisetsky, E. M., Lichtenstein, P., & Pedersen, N.L. (2010). Understanding the relation between anorexia nervosa and bulimia nervosa in a Swedish national twin sample. *Biological Psychiatry*, 67, 1, 71-77.
- Butler, A. C., Chapman, J. E., Forman, E. M., & Beck, A. T. (2006). The empirical status of cognitive-behavioural therapy: a review of meta-analyses. *Clinical psychology review*, 26(1), 17-31.
- Butryn, M.L., & Wadden, T.A. (2005). Treatment of overweight in children and adolescents: Does dieting increase the risk of eating disorders? *International Journal of eating Disorders*, 37, 4, 285-293.
- Button, E. J., Fransella, F., & Slade, P. D. (1977). A reappraisal of body perception disturbance in anorexia nervosa. *Psychological Medicine*, 7(02), 235-243.
- Button, E.J., Sonuga-Barke, E.J.S., Davies, J., & Thompson, M. (1996). A prospective study of self esteem in the prediction of eating problems in adolescent schoolgirls: questionnaire findings. *British Journal of Clinical Psychology*, 35, 193-203.

- Carter, J. C., Aime, A. A., & Mills, J. S. (2001). Assessment of bulimia nervosa: A comparison of interview and self-report questionnaire methods. *International Journal of Eating Disorders*, 30, 187–192.
- Carter, J.C., Blackmore, E., Sutandar-Pinnock, K., & Woodside, D.B. (2004). Relapse in anorexia nervosa: a survival analysis. *Psychological Medicine*, 34, 671-679.
- Case, L.K., Wilson, R.C., & Ramachandran, V.S. (2012). Diminished size-weight illusion in anorexia nervosa: evidence for visuo-proprioceptive integration deficit. *Experimental Brain Research*, 217, 1, 79-87.
- Cash, T. F., & Brown, T. A. (1987). Body image in anorexia nervosa and bulimia nervosa: A review of the literature. *Behaviour Modification*, 11, 487–521.
- Cash, T.F., & Deagle, E.A. (1997). The nature and extent of body-image disturbances in anorexia nervosa and bulimia nervosa: A meta-analysis. *International Journal of Eating Disorders*, 22, 107-126.
- Cash, T.F., & Smolak, L. (2011) Understanding body images: historical and contemporary perspectives. In T.F. Cash & L. Smolak (Eds.), *Body Image*,

Second Edition: A Handbook of Science, Practice, and Prevention. New York: Guilford Press.

Chan, Y.L., Leung, S.S.F., Lam, W.W.M., Peng, X.H., & Metreweli, C. (1998). Body fat estimation in children by magnetic resonance imaging, bioelectrical impedance, skinfold and body mass index: A pilot study. *Journal of Paediatrics and Child Health*, 34, 1, 22-28.

Channon, S., & De Silva, W. (1985). Psychological correlates of weight gain in patients with anorexia nervosa. *Journal of Psychiatric Research*, 19, 267-271.

Channon, S., De Silva, P., Hemsley, D., & Perkins, R. (1989). A controlled trial of cognitive-behavioural and behavioural treatment of anorexia nervosa. *Behaviour research and therapy*, 27(5), 529-535.

Cho, A., & Lee, J-L. (2013). Body dissatisfaction levels and gender differences in attentional biases toward idealized bodies. *Body Image*, 10, 95-102.

Claes, L., Bijttebier, P., Mitchell, J. E., de Zwaan, M., & Mueller, A. (2011). The relationship between compulsive buying, eating disorder symptoms, and temperament in a sample of female students. *Comprehensive Psychiatry*, 52(1), 50-55.

Clarke, T.K., Weiss, A.R.D., & Berrettini, W.H. (2012). The genetics of anorexia nervosa. *Clinical Pharmacology & Therapeutics*, 91, 2, 181-188.

Clausen, L. (2004). Time course of symptom remission in eating disorders. *International Journal of Eating Disorders*, 36, 3, 296-306.

Coetzee, V., Perrett, D.I., & Stephen, I.D. (2009). Facial adiposity: a cue to health? *Perception*, 38, 1700–1711.

Coetzee, V., Jingying, C., Perrett, D.I., & Stephen, I.D. (2010). Deciphering faces: quantifiable visual cues to weight. *Perception*, 39, 51–61.

Cohen-Tovée, E. M. (1993). Depressed mood and concern with weight and shape in normal young women. *International Journal of Eating Disorders*, 14(2), 223-227.

Cohn, L. D., Adler, N. E., Irwin, C. E., Millstein, S. G., Kegeles, S. M., & Stone, G. (1987). Body-figure preferences in male and female adolescents. *Journal of abnormal psychology*, 96(3), 276.

- Collier, D.A., Arranz, M.J., Li, T., Mupita, D., Brown, N., & Treasure, J. (1997). Association between 5-HT_{2A} gene promoter polymorphism and anorexia nervosa. *The Lancet*, 350, 9075, 412-412.
- Connan, F., Campbell, I.C., Katzman, M., Lightman, S.L., & Treasure, J. (2002). A neurodevelopmental model for anorexia nervosa. *Physiology and Behaviour*, 79, 1, 13-24.
- Cooper, P. J., & Taylor, M. J. (1988). Body image disturbance in bulimia nervosa. *British Journal of Psychiatry*, 153 (Suppl. 2), 32–36.
- Cooper, P.J., M.J. Taylor, Z. Cooper & C.G. Fairburn (1986). The development and validation of the Body Shape Questionnaire. *International Journal of Eating Disorders*, 6, 485-494.
- Cornelissen, P.L., Hancock, P.J.B., Kiviniemi, V., George, H.R., & Tovée, M.J. (2009a). Patterns of eye movements when male and female observers judge female attractiveness, body fat and waist-to-hip ratio. *Evol & Hum Behav*, 30, 417-428.
- Cornelissen, P.L., Tovée, M.J. and Bateson, M. (2009b). Patterns of subcutaneous fat deposition and the relationship between body mass index and waist-to-hip

ratio: Implications for models of physical attractiveness. *Journal of Theoretical Biology*, 256, 3, 343-350.

Cornelissen, P.L., Johns, A., & Tovée, M.J. (2013). Body size over-estimation in women with anorexia nervosa is not qualitatively different from female controls. *Body Image*, 10, 103-111.

Cornelissen, K. K., Bester, A., Cairns, P., Tovée, M. J., & Cornelissen, P. L. (2015). The influence of personal BMI on body size estimations and sensitivity to body size change in anorexia spectrum disorders. *Body image*, 13, 75-85.

Craig, A.D. (2009a). How do you feel—now? The anterior insula and human awareness. *Nat Rev Neurosci*, 10, 59-70.

Craig, A.D. (2009b). How do you feel? Interoception: the sense of the physiological condition of the body. *Nat Rev Neurosci*, 3, 655-66.

Crisp, A. H. (1997). Anorexia nervosa as flight from growth: Assessment and treatment based on the model. *Handbook of treatment for eating disorders*, 248-277.

Crisp, A. H., Hall, A., & Holland, A. J. (1985). Nature and nurture in anorexia nervosa: A study of 34 pairs of twins, one pair of triplets, and an adoptive family. *International Journal of Eating Disorders*, 4(1), 5-27.

Crisp, A.H., & Kalucy, R.S. (1974). Aspects of perceptual disorder in anorexia nervosa. *British Journal of Medical Psychology*, 47, 349-361.

Crisp, A. H., Harding, B., & McGuinness, B. (1974). Anorexia nervosa. Psychoneurotic characteristics of parents: Relationship to prognosis: A quantitative study. *Journal of Psychosomatic Research*, 18(3), 167-173.

Crisp, A. H., & Stonehill, E. (1971). Relation between aspects of nutritional disturbance and menstrual activity in primary anorexia nervosa. *BMJ*, 3(5767), 149-151.

Crossley, K.L., Cornelissen, P.L., & Tovée, M.J. (2012). What is an Attractive Body? Using an Interactive 3D Program to Create the Ideal Body for You and Your Partner. *PLoS One*, 7, e50601.

Dare, C. (1995). *Psychoanalytic Psychotherapy: Treatments of psychiatric disorders* 2nd ed. Washington: American Psychiatric Press.

- Dare, C., Eisler, I., Russell, G., Treasure, J., & Dodge, L. I. Z. (2001). Psychological therapies for adults with anorexia nervosa. *The British Journal of Psychiatry*, 178(3), 216-221.
- Davis, C., Fox, J., Cowles, M., Hastings, P., & Schwass, K. (1990). The functional role of exercise in the development of weight and diet concerns in women. *Journal of Psychosomatic Research*, 34(5), 563-574.
- Davis, C., Shuster, B., Blackmore, E., & Fox, J. (2004). Looking good—Family focus on appearance and the risk for eating disorders. *International Journal of Eating Disorders*, 35(2), 136-144.
- Deter, H. C., & Herzog, W. (1994). Anorexia nervosa in a long-term perspective: results of the Heidelberg-Mannheim Study. *Psychosomatic Medicine*, 56(1), 20-27.
- Dittmar, H., & Howard, S. (2004). Thin-ideal internalization and social comparison tendency as moderators of media models' impact on women's body-focused anxiety. *Journal of Social and Clinical Psychology*, 23(6), 768.

- Dohnt, H., & Tiggemann, M. (2006). The contribution of peer and media influences to the development of body satisfaction and self-esteem in young girls: a prospective study. *Developmental psychology*, 42(5), 929.
- Durnin, J., & Womersley, J. (1974). Body fat assessed from total body density and its estimation from skinfold thickness: measurements on 481 men and women aged from 16 to 72 years. *British Journal of Nutrition*, 32, 77-97.
- Eggert, J., Levendosky, A., & Klump, K. (2007). Relationships among attachment styles, personality characteristics, and disordered eating. *International Journal of Eating Disorders*, 40(2), 149.
- Ehrsson, H.H. (2007). The experimental induction of out-of-body experiences. *Science*, 317, 1048.
- Evans, C., & Dolan, B. (1993). Body Shape Questionnaire: derivation of shortened "alternate forms". *Int J Eat Disord*, 13, 315-321.
- Fairburn, C. G. (2008). *Cognitive behaviour therapy and eating disorders*. Guilford Press.

Fairburn, C. G., & Beglin, S. J. (1994). Assessment of eating disorders: Inter-view or self-report questionnaire? *International Journal of Eating Disorders*, 16,363–370.

Fairburn, C. G., & Cooper, P. J. (1989). Cognitive-behaviour therapy for psychiatric problems: a practical guide.

Fairburn, C. G., & Cooper, Z. (1993). The Eating Disorder Examination (12th ed.). In C. G. Fairburn, & G. T. Wilson (Eds.), *Binge eating: Nature, assessment and treatment* . New York: Guilford Press.

Fairburn, C.G., & Cooper, Z. (2011). Eating disorders, DSM-5 and clinical reality. *British Journal of Psychiatry*, 198, 1, 8-10.

Fairburn, C. G., Cooper, Z., & Cooper, P. J. (1986). The clinical features and maintenance of bulimia nervosa. *Handbook of eating disorders: Physiology, psychology and treatment of obesity, anorexia and bulimia*, 389-404.

Fairburn, C. G., Cooper, Z., Doll, H. A., O'Connor, M. E., Bohn, K., Hawker, D. M., & Palmer, R. L. (2009). Transdiagnostic cognitive-behavioural therapy for patients with eating disorders: a two-site trial with 60-week follow-up. *American Journal of Psychiatry*.

- Fairburn, C.G., Cooper, Z., Doll, H.A., O'Connor, M.E., Bohn, K., Hawker, D.M., Wales, J.A., & Palmer, R.L. (2009). Transdiagnostic cognitive-behavioural therapy for patients with eating disorders: A two-site trial with 60-week follow-up. *American Journal of Psychiatry*, 166, 3, 311-319.
- Fairburn, C.G., Cooper, Z., Doll, H.A., & Welch, S.L. (1999). Risk factors for anorexia nervosa - Three integrated case-control comparisons. *Archives of General Psychiatry*, 56, 5, 468-476.
- Fairburn, C.G., Cooper, Z., & Shafran, R. (2003). Cognitive behaviour therapy for eating disorders: A “transdiagnostic” theory and treatment. *Behaviour Research and Therapy*, 41, 509-528.
- Fairburn, C.G., & Harrison, P.J. (2003). Eating disorders. *Lancet*, 361, 407–16.
- Fairburn, C. G., Jones, R., Peveler, R. C., Carr, S. J., Solomon, R. A., O'Connor, M. E., & Hope, R. A. (1991). Three psychological treatments for bulimia nervosa: A comparative trial. *Archives of General Psychiatry*, 48(5), 463-469.
- Farrell, C., Lee, M., & Shafran, R. (2005). Assessment of body size estimation: a review. *European Eating Disorders Review*, 13, 75-88.

Farrell, C., Shafran, R., & Lee, M. (2006). Empirically evaluated treatments for body image disturbance: a review. *European Eating Disorders Review*, 14, 5, 289-300.

Favaro, A., Santonastaso, P., Manara, R., Bosello, R., Bommarito, G., Tenconi, E., & Di Salle, F. (2012). Disruption of visuospatial and somatosensory functional connectivity in anorexia nervosa. *Biological Psychiatry*, 72, 10, 864-870.

Fernández, F., Probst, M., Meermann, R., & Vandereycken, W. (1994). Body size estimation and body dissatisfaction in eating disorder patients and normal controls. *International Journal of Eating Disorders*, 16, 307–310.

Festinger, L. (1954). A theory of social comparison processes. *Human relations*, 7(2), 117-140.

Fladung, A.K., Grön, G., Grammer, K., Herrnberger, B., Schilly, E., Grasteit, S., Wolf, R.C., Walter, H., & von Wietersheim, J. (2010). A neural signature of anorexia nervosa in the ventral striatal reward system. *American Journal of Psychiatry*, 167, 206–212.

Freeman, R. J., Beach, B., Davis, R., & Solyom, L. (1985). The prediction of relapse in bulimia nervosa. *Journal of Psychiatric Research*, 19(2), 349-353.

- Freeman, R. J., Thomas, C. D., Solyom, L., & Miles, J. E. (1984). Body image disturbance in anorexia nervosa: A reexamination and a new technique. In P. L. Darby, P. E. Garfinkel & D. M. Garner (Eds), *Anorexia Nervosa: New Developments in Research*, pp. 117-127. New York: Liss.
- Gardner, R.M., & Bokenkamp, E.D. (1996). The role of sensory and nonsensory factors in body size estimations of eating disorder subjects. *Journal of Clinical Psychology*, 52, 3-15.
- Gardner, R.M., & Brown, D.L. (2014). Body size estimation in anorexia nervosa: A brief review of findings from 2003 through 2013. *Psychiatry Research*, 219, 3.
- Gardner, R.M., Brown, D.L., & Boice, R. (2012). Using amazon's mechanical turk website to measure accuracy of body size estimation and body dissatisfaction. *Body image*, 9, 532-534.
- Gardner, R. M., Friedman, B. N., & Jackson, N. A. (1999). Body size estimations, body dissatisfaction, and ideal size preferences in children six through thirteen. *Journal of youth and adolescence*, 28(5), 603-618.

Gardner, R.M., Jones, L.C., & Bokenkamp, E.D. (1995). Comparison of three psychophysical techniques for estimating body-size perception. *Perceptual and Motor Skills*, 80, 1379-1390.

Gardner, R.M., & Moncrieff, C. (1988). Body image distortion in anorexics as a non-sensory phenomenon: A signal detection approach. *Journal of Clinical Psychology*, 44, 101-107.

Garfinkel, P.E., Kennedy, S.H., & Kaplan, A.S. (1995). Views on classification and diagnosis of eating disorders. *Canadian Journal of Psychiatry - Revue Canadienne de Psychiatrie*, 40, 8, 445-456.

Garner, D.M. (1993). Pathogenesis of anorexia nervosa. *The Lancet*, 341, 1631-1640.

Garner, D. M., & Garfinkel, P. E. (1980). Socio-cultural factors in the development of anorexia nervosa. *Psychological medicine*, 10(04), 647-656.

Garner, D. M., & Garfinkel, P. E. (1982). Body image in anorexia nervosa: Measurement, theory and clinical implications. *The International Journal of Psychiatry in Medicine*, 11(3), 263-284.

- Garner, D. M., Garfinkel, P. E., Stancer, H. C., & Moldofsky, H. (1976). Body image disturbances in anorexia nervosa and obesity. *Psychosomatic Medicine*, 38, 327–336.
- Garner, D. M., Olmstead, M. P., & Polivy, J. (1982). Development and validation of a multidimensional eating disorder inventory for anorexia nervosa and bulimia. *International journal of eating disorders*, 2(2), 15-34.
- Garner, D. M., Vitousek, K. M., & Pike, K. M. (1997). Cognitive-behavioural therapy for anorexia nervosa. *Handbook of treatment for eating disorders*, 2, 121-134.
- Gaudio, S., & Quattrocchi, C.C. (2012). Neural basis of a multidimensional model of body image distortion in anorexia nervosa. *Neuroscience and Neurobehavioural Reviews*, 36, 1839-1847.
- Geller, J., Johnston, C., Madsen, K., Goldner, E.M., Remick, R.A., & Birmingham, C.L. (1998). Shape and weight-based self-esteem and the eating disorders. *International Journal of Eating Disorders*, 24, 285–98
- George, H.R., Cornelissen, P.L., Hancock, P.J., Kiviniemi, V.V., & Tovée, M.J. (2011). Differences in eye-movement patterns between anorexic and control

observers when judging body size and attractiveness. *Br J Psychol*, 102, 340-354.

Gershon, E. S., Schreiber, J. L., Hamovit, J. R., Dibble, E. D., Kaye, W., Nurnberger, J. I., ... & Ebert, M. (1984). Clinical findings in patients with anorexia nervosa and affective illness in their relatives. *The American journal of psychiatry*, 141(11), 1419-1422.

Gescheider, G. A. (1997). *Psychophysics: The Fundamentals (3rd ed.)*. New Jersey: Lawrence Erlbaum.

Gila, A., Castro, J., and Toro, J. (1998). Subjective body-image dimensions in normal and anorexic adolescents. *British Journal of Medical Psychology*, 71, 175-184.

Gila, A., Castro, J., Cesena, J., and Toro, J. (2005). Anorexia nervosa in male adolescents: body image, eating attitudes and psychological traits. *Journal of Adolescent Health*, 36, 221-226.

Ginis, K.A.M., & Bassett, R.L. (2011). Exercise and changes in body image. In T.F. Cash & L. Smolak (Eds.), *Body Image: A Handbook of Science, Practice, and Prevention* (pp. 415-423). New York: Guilford Press.

Gledhill, L.J. (2015). *Factors that Modulate Preference for Body Size and Shape and the Effect of Experience* (Doctoral dissertation). Retrieved from the Database of Institute of Neuroscience, Newcastle University (Retrieval date 2.6.2016).

Godart, N.T., Flament, M.F., Lecrubier, Y., & Jeammet, P. (2000). Anxiety disorders in anorexia nervosa and bulimia nervosa: co-morbidity and chronology of appearance. *European Psychiatry*, 15, 38–45.

Goldbloom, D. S., & Olmstead, M. P. (1993). Pharmacotherapy of bulimia nervosa with fluoxetine. *Am. J. Psychiatry*, 150, 770-774.

Grabe, S., Ward, L.M., & Hyde, J.S. (2008). The role of the media in body image concerns among women: a meta-analysis of experimental and correlational studies. *Psychol Bull*, 134, 3, 460-76.

Gray, J., Yeo, G. S., Cox, J. J., Morton, J., Adlam, A. L. R., Keogh, J. M., ... & Farooqi, I.S. (2006). Hyperphagia, severe obesity, impaired cognitive function, and hyperactivity associated with functional loss of one copy of the brain-derived neurotrophic factor (BDNF) gene. *Diabetes*, 55(12), 3366-3371.

Green, D.M., & Swets, J.A. (1966). *Signal detection theory and psychophysics*. New York: Wiley.

Grilo, C. M., Masheb, R. M., & Wilson, G. T. (2001). A comparison of different methods for assessing the features of eating disorders in patients with binge eating disorder. *Journal of Consulting and Clinical Psychology*, 69, 317–322.

Groesz, L. M., Levine, M. P., & Murnen, S. K. (2002). The effect of experimental presentation of thin media images on body satisfaction: A meta-analytic review. *International Journal of Eating Disorders*, 31(1), 1-16.

Grondin, S. (2012). Violation of the scalar property for time perception between 1 and 2 seconds: Evidence from interval discrimination, reproduction and categorization. *Journal of Experimental Psychology: Human Perception and Performance*, 38, 4, 880–890.

Gull, W. W. (1873). Clinical society, Friday, October 24th, 1873. Anorexia hysterica (apepsia hysterica). *British Medical Journal*, 2, 527-529.

Gustavson, C. R., Gustavson, J. C., Pumariega, A. J., Reinartz, D. E., Gustavson, R. D. A. R., Pappas, T., & McCaul, K. E. V. I. N. (1990). Body-image distortion among male and female college and high school students, and eating-disordered patients. *Perceptual and motor skills*, 71(3), 1003-1010.

- Halmi, K. A., Agras, W. S., Crow, S., Mitchell, J., Wilson, G. T., Bryson, S. W., & Kraemer, H. C. (2005). Predictors of treatment acceptance and completion in anorexia nervosa: implications for future study designs. *Archives of General Psychiatry*, 62(7), 776-781.
- Halmi, K. A., Sunday, S. R., Strober, M., Kaplan, A., Woodside, D. B., Fichter, M., & Kaye, W. H. (2000). Perfectionism in anorexia nervosa: variation by clinical subtype, obsessionality, and pathological eating behaviour. *American Journal of Psychiatry*.
- Hamilton, L. H., Brooks-Gunn, J., & Warren, M. P. (1985). Sociocultural influences on eating disorders in professional female ballet dancers. *International Journal of Eating Disorders*, 4(4), 465-477.
- Hancock, P.J.B. (2000). Evolving faces from principal components. *Behaviour Research Methods, Instruments and Computers*, 32, 327-333.
- Harrison, K., & Cantor, J. (1997). The relationship between media consumption and eating disorders. *Journal of Communication*, 47(1), 40-67.

Hasler, N., Stoll, C., Sunkel, M., Rosenhahn, B., & Seidel, H. P. (2009). A statistical model of human pose and body shape. *Computer Graphics Forum*, 28, 337–346.

Haworth-Hoeppner, S. (2000). The critical shapes of body image: The role of culture and family in the production of eating disorders. *Journal of Marriage and Family*, 62(1), 212-227.

Hay, P.J., Claudino, A.M., Touyz, S., & Abd Elbaky, G. (2015) Individual psychological therapy in the outpatient treatment of adults with anorexia nervosa. *Cochrane Database of Systematic Reviews* 7, CD003909. DOI:10.1002/14651858.CD003909.pub2.

Health Survey for England (2008). National Centre for Social Research and University College London. Department of Epidemiology and Public Health. UK Data Archive, Colchester, Essex.

Health Survey for England (2012). National Centre for Social Research and University College London. Department of Epidemiology and Public Health. UK Data Archive, Colchester, Essex, UK.

Heatherton, T. F., Mahamedi, F., Striepe, M., Field, A. E., & Keel, P. (1997). A 10-year longitudinal study of body weight, dieting, and eating disorder symptoms. *Journal of abnormal psychology, 106*(1), 117.

Heatherton, T.F., & Polivy, J. (1992). Chronic dieting and eating disorders: a spiral model. In *The Etiology of Bulimia: The Individual and Familial Context*, ed. J. Crowther, S.E. Hobfall, M.A.P. Stephens, D.L. Tennenbaum. Washington, DC: Hemisphere.

Hewig, J., Cooper, S., Trippe, R.H., Hecht, H., Straube, T., & Miltner, W.H.R. (2008). Drive for thinness and attention toward specific body parts in a nonclinical sample. *Psychosom Med, 70*, 729–736.

Hodzic, A., Muckli, L., Singer, W., & Stirn, A. (2009). Cortical responses to self and others. *Human Brain Mapping, 30*, 951–962.

Hoek, H. W. (2006). Incidence, prevalence and mortality of anorexia nervosa and other eating disorders. *Current Opinion in Psychiatry, 19*, 4, 389-394.

Hoek, H. W., Bartelds, A. I., Bosveld, J. J., & van der Graaf, Y. (1995). Impact of urbanization on detection rates of eating disorders. *The American journal of psychiatry, 152*(9), 1272.

- Hoek, H. W., & Van Hoeken, D. (2003). Review of the prevalence and incidence of eating disorders. *International Journal of eating disorders*, 34(4), 383-396.
- Horndasch, S., Kratz, O., Holczinger, A., Heinrich, H., Hönig, F., Nöth, E., & Moll, G.H. (2012). "Looks do matter"--visual attentional biases in adolescent girls with eating disorders viewing body images. *Psychiatry Res*, 198, 321-3.
- Insel, T., Cuthbert, B., Garvey, M., Heinssen, R., Pine, D.S., Quinn, K., & Wang, P. (2010). Research Domain Criteria (RDoC): Toward a new classification framework for research on mental disorders. *American Journal of Psychiatry*, 167, 7, 748-751.
- Irving, L. M., & Berel, S. R. (2001). Comparison of media-literacy programs to strengthen collage women's resistance to media images [electronic version]. *Psychology of Women Quarterly*, 25, 103-111.
- Jackson, T. D., Grilo, C. M., & Masheb, R. M. (2000). Teasing history, onset of obesity, current eating disorder psychopathology, body dissatisfaction, and psychological functioning in binge eating disorder. *Obesity Research*, 8(6), 451-458.

Janelle, C.M., Hausenblas, H.A., Ellis, R.E., Coombes, S.A., & Duley, A.R. (2009).

The time course of attentional allocation while women high and low in body dissatisfaction view self and model physiques. *Psychol Health*, 24, 351–366.

Jansen, A., Nederkoorn, C., and Mulkens, S. (2005). Selective visual attention for ugly and beautiful body parts in eating disorders. *Behavioural Research and Therapy*, 43(2), 183-196.

Jarry, J.L., & Cash, T.F. (2011). Cognitive-behavioural approaches to body image change. In T.F. Cash & L. Smolak (Eds.), *Body Image: A Handbook of Science, Practice, and Prevention* (pp. 415-423). New York: Guilford Press.

Johnson, F., & Wardle, J. (2005). Dietary restraint, body dissatisfaction, and psychological distress: A prospective analysis. *Journal of Abnormal Psychology*, 114, 1, 119-125.

Joos, A., Klöppel, S., Hartmann, A., Glauche, V., Tüscher, O., Perlov, E., & van Elst, L.T. (2010). Voxel-based morphometry in eating disorders: Correlation of psychopathology with grey matter volume. *Psychiatry Res*, 182, 146–151.

Katzman, D.K., Lambe, E.K., Mikulis, DJ, Ridgely, J.N., Goldbloom, D.S., & Zipursky, R.B. (1996). Cerebral gray matter and white matter volume deficits

in adolescent girls with anorexia nervosa. *Journal of Pediatrics*, 129, 6, 794-803.

Kaye, W.H., Bailer, U.F., Frank, G.K., Wagner, A., & Henry, S.E. (2005). Brain imaging of Serotonin after recovery from anorexia and bulimia nervosa. *Physiology and Behaviour*, 86, 15-17.

Kaye, W.H., Fudge, J.L., & Paulus, M. (2009). New insights into symptoms and neurocircuit function of anorexia nervosa. *Nature Reviews Neuroscience*, 10, 573-584.

Keating, C. (2010). Theoretical perspective on anorexia nervosa: The conflict of reward. *Neuroscience and Behavioural Reviews*, 34, 1, 73-79.

Keel, P. K., Brown, T. A., Holland, L. A., & Bodell, L.P. (2012). Empirical Classification of Eating Disorders. *Annual Review of Clinical Psychology*, 8, 381-404.

Keel, P. K., Heatherton, T. F., Harnden, J. L., & Hornig, C. D. (1997). Mothers, fathers, and daughters: Dieting and disordered eating. *Eating Disorders*, 5(3), 216-228.

Keel, P. K., & Klump, K. L. (2003). Are eating disorders culture-bound syndromes? Implications for conceptualizing their etiology. *Psychological bulletin*, 129(5), 747.

Keim, N.L., Stern, J.S., & Havel, P.J. (1998). Relation between circulating leptin concentrations and appetite during a prolonged, moderate energy deficit in women. *American Journal of Clinical Nutrition*, 68, 4, 794-801.

Ketel, I.J.G., Volman, M.N.M., Seidell, J.C., Stehouwer, C.D.A., Twisk, J.W., & Lambalk, C.B. (2007). Superiority of skinfold measurements and waist over waist-to-hip ratio for determination of body fat distribution in a population-based cohort of Caucasian Dutch adults. *European Journal of Endocrinology*, 156, 6, 655-661.

Key, A., George, C.L., Beattie, D., Stammers, K., Lacey, H., & Waller, G. (2002). Body image treatment within an inpatient program for anorexia nervosa: the role of mirror exposure in the desensitization process. *International Journal of Eating Disorders*, 31, 2, 185-190.

Keys, A., Brožek, J., Henschel, A., Mickelsen, O., & Taylor, H. L. (1950). *The biology of human starvation*.

- Keys, A., Vivanco, F., Minon, J., Keys, M., & Mendoza, H. (1954). Studies on the diet, body fatness and serum cholesterol in Madrid, Spain. *Metabolism Clinical and Experimental*, 3, 3, 195-212.
- King, J.A., Geisler, D., Ritschel, F., Boehm, I., Seidel, M., Roschinski, B., ... & Ehrlich, S. (2014). Global cortical thinning in acute anorexia nervosa normalizes following long-term weight restoration. *Biol Psychiatry*, 77, 624-632.
- Klesges, R. C. (1983). An analysis of body image distortions in a nonpatient population. *International Journal of Eating Disorders*, 2(2), 35-41.
- Klump, K.L., Miller, K.B., Keel, P.K., McGue, M., & Iacono, W.G. (2001). Genetic and environmental influences on anorexia nervosa syndromes in a population-based twin sample. *Psychological Medicine*, 31, 737-740.
- Knauss, C., Paxton, S. J., & Alsaker, F. D. (2007). Relationships amongst body dissatisfaction, internalisation of the media body ideal and perceived pressure from media in adolescent girls and boys. *Body Image*, 4(4), 353-360.

- Kohrt, W.M. (1998). Preliminary evidence that DEXA provides an accurate assessment of body composition. *Journal of Applied Physiology*, 84, 1, 372-377.
- Kortegaard, L.S., Hoerder, K., Joergensen, J., Gillberg, C., & Kyvik, K.O. (2001). A preliminary population-based twin study of self-reported eating disorder. *Psychological Medicine*, 31, 361–365.
- Kuchler, F., & Variyam, J.N. (2003). Mistakes were made: misperception as a barrier to reducing overweight. *International Journal of Obesity*, 27, 856–886.
- Kulbartz-Klatt, Y.J., Florin, I., & Pook, M. (1999). Bulimia nervosa: mood changes do have an impact on body width estimation. *British Journal of Clinical Psychology*, 38, 279–87.
- Kuskowska-Wolk, A., & Rössner, S. (1989). The “true” prevalence of obesity: a comparison of objective weight and height measures versus self-reported and calibrated data. *Scandinavian Journal of Primary Health Care*, 7, 79-82.
- Lautenbacher, S., Kraehe, N., & Krieg, J.C. (1997) Perception of body size and body satisfaction in recovered anorexic women: Comparison with restrained and unrestrained eaters. *Perceptual and Motor Skills*, 84, 3, 1331-1342.

- Lawrence, A.D., Dowson, J., & Foxall, G.L. (2003). Impaired visual discrimination learning in anorexia nervosa. *Appetite*, 40, 1, 85-89.
- Leahy, S., O'Neill, C., Sohun, R., & Jakeman, P. (2012). A comparison of dual energy X-ray absorptiometry and bioelectrical impedance analysis to measure total and segmental body composition in healthy young adults. *European Journal of Applied Physiology*, 112, 2, 589-595.
- Lee, Y. H., Rhee, M. K., Park, S. H., Sohn, C. H., Chung, Y. C., Hong, S. K., ..., & Olivos, M. (1998). Epidemiology of eating disordered symptoms in the Korean general population using a Korean version of the Eating Attitudes Test. *Eating and Weight Disorders-Studies on Anorexia, Bulimia and Obesity*, 3(4), 153-161.
- Lehoux, P.M., Steiger, H., & Jabalpurawa, S. (2000). State/trait distinctions in bulimic syndromes. *International Journal of Eating Disorders*, 27, 36-42
- Lemieux, S., Prudhomme, D., Bouchard, C., Tremblay, A., & Despres, J.P. (1993). Sex-differences in the relation of visceral adipose-tissue accumulation to total-body fatness. *American Journal of Clinical Nutrition*, 58, 4, 463-467.

- Leopold, D. A., O'Toole, A. J., Vetter, T., & Blanz, V. (2001). Prototype-referenced shape encoding revealed by high-level after effects. *Nature Neuroscience*, 4, 89–94.
- Levi, D.M., Klein, A.A., & Aitsebaomo, A.P. (1985). Vernier acuity, crowding and cortical magnification. *Vis Research*, 25, 963–977.
- Levine, M.P., & Murnen, S.K. (2009). “Everybody knows that mass media are/are not [pick one] a cause of eating disorders”: A critical review of evidence for a causal link between media, negative body image, and disordered eating in females. *Journal of Social and Clinical Psychology*, 28, 1, 9-42.
- Levine, M. P., & Smolak, L. (2001). Primary prevention of body image disturbances and disordered eating in childhood and early adolescence. *Body image, eating disorders, and obesity in youth: Assessment, prevention, and treatment*, 237-260.
- Ley, C.J., Lees, B., & Stevenson, J.C. (1992). Sex-associated and menopause-associated changes in body-fat distribution. *American Journal of Clinical Nutrition*, 55, 5, 950-954.

- Lohman, T.G., Caballero, B., Himes, J.H., Davis, C.E., Stewart, D., Houtkooper, L., & Stephenson, L. (2000). Estimation of body fat from anthropometry and bioelectrical impedance in Native American children. *International Journal of Obesity*, 24, 8, 982-988.
- Luce, K. H., & Crowther, J. H. (1999). The reliability of the Eating Disorder Examination-Self-Report Questionnaire version (EDE-Q). *International Journal of Eating Disorders*, 25, 349–351.
- Luce, K.H., Crowther, J.H., & Pole, M. (2008). Eating Disorder Examination Questionnaire (EDE-Q): Norms for Undergraduate Women. *Int J Eat Disord*, 41, 3, 273–276.
- Madsen, S. K., Bohon, C., & Feusner, J. D. (2013). Visual processing in anorexia nervosa and body dysmorphic disorder: Similarities, differences, and future research directions. *Journal of Psychiatric Research*, 47, 10, 1483-1491.
- Marcoa, J.H., Perpiñá, C., & Botellac, C. (2013). Effectiveness of cognitive behavioural therapy supported by virtual reality in the treatment of body image in eating disorders: One year follow-up. *Psychiatry Research*, 209, 3, 619-625.

Martin, K.A., & Lichtenberger, C.M. (2002). Fitness enhancement and changes in body image. In T. F. Cash & T. Pruzinsky (Eds.), *A handbook of theory, research, and clinical practice* (pp. 414-421). New York: The Guilford Press.

Martínez-González, M. A., Gual, P., Lahortiga, F., Alonso, Y., de Irala-Estévez, J., & Cervera, S. (2003). Parental factors, mass media influences, and the onset of eating disorders in a prospective population-based cohort. *Pediatrics*, *111*(2), 315-320.

Maximova, K., McGrath, J.J., Barnett, T., O'Loughlin, J., Paradis, G., & Lambert, M. (2008). Do you see what I see? Weight status misperception and exposure to obesity among children and adolescents. *International Journal of Obesity*, *32*, 1008-1015.

McCabe, M. P., & Ricciardelli, L. A. (2001). Parent, peer, and media influences on body image and strategies to both increase and decrease body size among adolescent boys and girls. *Adolescence*, *36*(142), 225.

McIntosh, V. V., Jordan, J., Luty, S. E., Carter, F. A., McKenzie, J. M., Bulik, C. M., & Joyce, P. R. (2006). Specialist supportive clinical management for anorexia nervosa. *International Journal of Eating Disorders*, *39*(8), 625-632.

- Meermann, R. (1983). Experimental investigation of disturbances in body image estimation in anorexia nervosa patients, and ballet and gymnastics pupils. *International Journal of Eating Disorders*, 2, 91–100.
- Mitchell, J. E., & Crow, S. (2006). Medical complications of anorexia nervosa and bulimia nervosa. *Current Opinion in Psychiatry*, 19, 4, 438-443.
- Mohr, H.M., Zimmermann, J., Roder, C., Lenz, C., Overbeck, G., & Grabhorn, R. (2010). Separating two components of body image in anorexia nervosa using fMRI. *Psychological Medicine*, 40, 1519–1529.
- Mond, J. M., Hay, P. J., Rodgers, B., Owen, C., & Beumont, P. J. V. (2004). Validity of the Eating Disorder Examination Questionnaire (EDE-Q) in screening for eating disorders in community samples. *Behaviour Research and Therapy*, 42, 551–567.
- Mond, J.M., Hay, P.J., Rodgers, B., & Owen, C. (2006). Eating Disorder Examination Questionnaire (EDE-Q): Norms for young adult women. *Behav Res Ther*. 44, 53–62.
- Moschos, M.M., Gonidakis, F., Varsou, E., Markopoulos, I., Rouvas, A., Ladas, I., & Papadimitriou, G.N. (2011). Anatomical and functional impairment of the

retina and optic nerve in patients with anorexia nervosa without vision loss.
British Journal of Ophthalmology, 95, 8, 1128-1133.

Murnen, S. K., Smolak, L., Mills, J. A., & Good, L. (2003). Thin, sexy women and strong, muscular men: Grade-school children's responses to objectified images of women and men. *Sex Roles*, 49(9-10), 427-437.

Mussap, A. J., McCabe, M. P., & Ricciardelli, L. A. (2008). Implications of accuracy, sensitivity, and variability of body size estimations to disordered eating. *Body Image*, 5, 80–90.

Nasser, M. (1986). Comparative study of the prevalence of abnormal eating attitudes among Arab female students of both London and Cairo universities.
Psychological medicine, 16(3), 621-625.

Nelson, M.E., Fiatarone, M.A., Layne, J.E., Trice, I., Economos, C.D., Fielding, R.A., & Evans, W.J. (1996). Analysis of body-composition techniques and models for detecting change in soft tissue with strength training. *American Journal of Clinical Nutrition*, 63, 5, 678-686.

- Norris, M.L., Boydell, K.M., Pinhas, L., & Katzman, D.K. (2006). Ana and the internet: A review of pro-anorexia websites. *International Journal of Eating Disorders*, 39, 443-447.
- Nunn, K., Frampton, I., Gordon, I., & Lask, B. (2008). The fault is not in her parents but in her insula — a neurobiological hypothesis of anorexia nervosa. *Eur Eat Disord Rev*, 16, 355-60.
- O'Dea, J. (2004). Evidence for a self-esteem approach in the prevention of body image and eating problems among children and adolescents. *Eating Disorders*, 12, 225-239.
- Pallanti, S., Quercioli, L., Zaccara, G., Ramacciotti, A.B., & Arnetoli, G. (1998). Eye movement abnormalities in anorexia nervosa. *Psychiatry Res*, 78, 59–70.
- Park, R. J., Godier, L. R., & Cowdrey, F. A. (2014). Hungry for reward: How can neuroscience inform the development of treatment for anorexia nervosa? *Behaviour Research and Therapy*, 62 Special Issue, 47-59.
- Penner, L. A., Thompson, J. K., & Coovert, D. L. (1991). Size overestimation among anorexics: Much ado about very little?. *Journal of abnormal psychology*, 100(1), 90.

Penton-Voak, I.S., Bate, H., Lewis, G.H., & Munafò, M.R. (2012). Effects of emotion perception training on mood in undergraduate students: A randomized controlled trial. *The British Journal of Psychiatry*, 201, 71-2.

Penton-Voak, I.S., Thomas, J., Gage, S.H., McMurrin, M., McDonald, S., & Munafò, M.R. (2013). Increasing recognition of happiness in ambiguous facial expressions reduces anger and aggressive behaviour. *Psychological Science*, 24, 688-97.

Perrett, D.I., Burt, D.M., Penton-Voak, I.S., Lee, K.J., Rowland, D.A., & Edwards, R. (1999). Symmetry and human facial attractiveness. *Evolution and Human Behaviour*, 20, 5, 295-307.

Phillipou, A., Rossell, S.L., Castle, D.J., Gurvich, C., & Abel, L.A. (2014). Square wave jerks and anxiety as distinctive biomarkers for anorexia nervosa. *Invest Ophthalmol Vis Sci*, 55, 8366–8370.

Pike, K.M. (1998). Long-term course of anorexia nervosa: Response, relapse, remission, and recovery. *Clinical Psychology Review*, 18, 4, 447-475.

Piryankova, I. V., Wong, H. Y., Linkenauger, S. A., Stinson, C., Longo, M. R., Bülthoff, H. H., & Mohler, B. J. (2014). Owning an overweight or underweight

body: distinguishing the physical, experienced and virtual body. *PloS one*, 9(8), e103428.

Podar, I., Hannus, A., & Allik, J. (1999). Personality and affectivity characteristics associated with eating disorders: A comparison of eating disordered, weight-preoccupied, and normal samples. *Journal of personality assessment*, 73(1), 133-147.

Polivy, J., & Herman, C.P. (1985). Dieting and bingeing: a causal analysis. *American Psychologist*, 40, 193–201.

Polivy, J., & Herman, C.P. (2002). Causes of eating disorders. *Annual Review of Psychology*, 53, 187-213.

Pollice, C., Kaye, W. H., Greeno, C. G., & Weltzin, T. E. (1997). Relationship of depression, anxiety, and obsessionality to state of illness in anorexia nervosa. *International Journal of Eating Disorders*, 21(4), 367-376.

Poulton, E.C. (1989). *Bias in Quantifying Judgements*. Erlbaum, Hove, UK.

- Power, M. L., & Schulkin, J. (2008). Sex differences in fat storage, fat metabolism, and the health risks from obesity: possible evolutionary origins. *British Journal of Nutrition*, 99, 5, 931-940.
- Preston, C., & Ehrsson, H. H. (2014). Illusory changes in body size modulate body satisfaction in a way that is related to non-clinical eating disorder psychopathology. *Plos One*, 9, 1: e85773
- Probst, M., Vandereycken, W., Van Coppenolle, H., & Pieters, G. (1995). Body size estimation in eating disorder patients: Testing the video distortion method on a life-size screen. *Behaviour Research and Therapy*, 33, 985-990.
- Probst, M., Vandereycken, W., & Van Coppenolle, H. (1997). Body-size estimation in eating disorders using video distortion on a life-size screen. *Psychotherapy and Psychosomatics*, 66, 87-91.
- Rankinen, T., Kim, S.Y., Perusse, L., Despres, J.P., & Bouchard, C. (1999). The prediction of abdominal visceral fat level from body composition and anthropometry: ROC analysis. *International Journal of Obesity*, 23, 8, 801-809.

- Ransom, D.C., La Guardia, J.G., Woody, E.Z., & Boyd, J.L. (2010). Interpersonal interactions on online forums addressing eating concerns. *International Journal of Eating Disorders*, 43, 161-170.
- Rhodes, G., Jeffery, L., Boeing, A., & Calder, A.J. (2013). Visual coding of human bodies: perceptual aftereffects reveal norm-based, opponent coding of body identity. *Journal of Experimental Psychology: Human Perception and Performance*, 39, 2, 313-317.
- Ribasés, M., Gratacòs, M., Fernández-Aranda, F., Bellodi, L., Boni, C., Anderluh, M., & Estivill, X. (2005). Association of BDNF with restricting anorexia nervosa and minimum body mass index: a family-based association study of eight European populations. *European Journal of Human Genetics*, 13(4), 428-434.
- Ricciardelli, L.A., & McCabe, M.P. (2001). Dietary restraint and negative affect as mediators of body dissatisfaction and bulimic behaviour in adolescent girls and boys. *Behaviour Research and Therapy*, 39, 11, 1317-1328.
- Riva, G., Bacchetta, M., Baruffi, M., & Molinari, E. (2002). Virtual-reality based multidimensional therapy for the treatment of body image disturbances in

binge eating disorders: a preliminary controlled study. *IEEE Transactions on Information Technology in Biomedicine*, 6, 224-234.

Robinson, E., & Kirkham, T.C. (2013). Is he a healthy weight? Exposure to obesity changes perception of the weight status of others. *International Journal of Obesity*, 38, 663-667.

Roefs, A., Jansen, A., Moresi, S., Willems, P., van Grootel, S., & van der Borgh, A. (2008). Looking good. BMI, attractiveness bias and visual attention. *Appetite*, 51(3), 552-555.

Romero-Corral, A., Somers, V.K., Sierra-Johnson, J., Thomas, R.J., Collazo-Clavell, M.L., Korinek, J., & Lopez-Jimenez, F. (2008). Accuracy of body mass index in diagnosing obesity in the adult general population. *International Journal of Obesity*, 32, 6, 959-966.

Rosen, J. C. (1996). Body image assessment and treatment in controlled studies of eating disorders. *International Journal of Eating Disorders*, 20(4), 331-343.

Rosen, J. C., Cado, S., Silberg, N. T., Srebnik, D., & Wendt, S. (1990). Cognitive behaviour therapy with and without size perception training for women with body image disturbance. *Behaviour Therapy*, 21(4), 481-498.

- Rosen, J.C., Jones, A., Ramirez, E., & Waxman, S. (1996). Body Shape Questionnaire: Studies of validity and reliability. *International Journal of Eating Disorders*, 20, 315–319.
- Rosen, J. C., Orosan, P., & Reiter, J. (1996). Cognitive behaviour therapy for negative body image in obese women. *Behaviour therapy*, 26(1), 25-42.
- Rosenberg, M. (1965). *Society and the adolescent self-image*. Princeton, NJ: Princeton University Press.
- Russell, G. F. M. (1979). Bulimia nervosa: An ominous variant of anorexia nervosa. *Psychological Medicine*, 9, 429–448.
- Sachdev, P., Mondraty, N., Wen, W., & Gulliford, K. (2008). Brains of anorexia nervosa patients process self-images differently from non-self-images: an fMRI study. *Neuropsychologia*, 46, 2161–2168.
- Santel, S., Baving, L., Krauel, K., Münte, T., & Rotte, M. (2006). Hunger and satiety in anorexia nervosa: fMRI during cognitive processing of food pictures. *Brain Research*, 1114, 138–148.

Schmidt, U., Oldershaw, A., Jichi, F., Sternheim, L., Startup, H., McIntosh, V., & Landau, S. (2012). Out-patient psychological therapies for adults with anorexia nervosa: randomised controlled trial. *The British Journal of Psychiatry*, 201(5), 392-399.

Schultz, W. (2004). Neural coding of basic reward terms of animal learning theory, game theory, microeconomics and behavioural ecology. *Science*, 14, 139–147.

Secord, P. F., & Jourard, S. (1953). The appraisal of body cathexis: Body cathexis and the self. *Journal of Consulting and Clinical Psychology*, 17, 343–347.

Shafran, R., & Fairburn, C. G. (2002). A new ecologically valid method to assess body size estimation and body size dissatisfaction. *International Journal of Eating Disorders*, 32, 458–465.

Shapiro, J.R., Berkman, N.D., Brownley, K.A., Sedway, J.A., Lohr, K.N., & Bulik, C.M. (2007). Bulimia nervosa treatment: A systematic review of randomized controlled trials. *International Journal of Eating Disorders*, 40, 4, 321-336.

Sheldon, W. H., Stevens, S. S., & Tucker, W. B. (1940). *The varieties of human physique*. New York, NY: Harper & Brothers.

Singh, D., & Singh, D. (2011). Shape and Significance of Feminine Beauty: An Evolutionary Perspective. *Sex Roles*, 64, 723-731.

Skrzypek, S., Wehmeier, P.M., & Remschmidt, H. (2001). Body image assessment using body size estimation in recent studies on anorexia nervosa. A brief review. *European Child & Adolescent Psychiatry*, 10, 215-221.

Slade, P., & Russell, G. (1973). Awareness of body dimensions in anorexia nervosa: cross-sectional and longitudinal studies. *Psychological Medicine*, 3, 188-199.

Slade, P. D. (1988). Body image in anorexia nervosa. *British Journal of Psychiatry*, 153(Suppl. 2), 20-22.

Smeets, M. A., Smit, F., Panhuysen, G. E., & Ingleby, J. D. (1997). The influence of methodological differences on the outcome of body size estimation studies in anorexia nervosa. *British Journal of Clinical Psychology*, 36(2), 263-277.

- Smeets, M.A.M. (1999). Body size categorization in anorexia nervosa using a morphing instrument. *International Journal of Eating Disorders*, 25, 4, 451-455.
- Smeets, M.A.M., Ingleby J.D., Hoek, H.W., & Panhuysen, G.E.M. (1999). Body size perception in anorexia nervosa: a signal detection approach. *Journal of Psychosomatic Research*, 46, 5, 465-477.
- Smith, K.L., Tovée, M.J., Hancock, P., Cox, M., & Cornelissen, P.L. (2007). An analysis of body shape attractiveness based on image statistics: evidence for a dissociation between expressions of preference and shape discrimination. *Visual Cognition*, 15(7), 1-27.
- Smolak, L. (2004). Body image in children and adolescents: where do we go from here? *Body image*, 1(1), 15-28.
- Smolak, L., & Levine, M. P. (2001). Body image in children. *Body image, eating disorders, and obesity in youth: Assessment, prevention, and treatment*, 41-66.
- Steiger, H., Leung, F.Y.K., Puentes-Neuman, G., & Gottheil, N. (1992). Psychological profiles of adolescent girls with varying degrees of eating and mood disturbances. *International Journal of Eating Disorders*, 11, 121-131.

- Steinglass, J., & Walsh, B.T. (2006). Habit learning and anorexia nervosa: A cognitive neuroscience hypothesis. *International Journal of Eating Disorders*, 39, 4, 267-275.
- Stewart, A., Crockett, P., Nevill, A., & Benson, P. (2014). Somatotype: a more sophisticated approach to body image work with eating disorder sufferers. *Advances in Eating Disorders: Theory, Research and Practice*, 2:2, 125-135.
- Stewart, A.D., Klein, S., Young, J., Simpson, S., Lee, A.J., Harrild, K., & Benson, P.J. (2012). Body image, shape, and volumetric assessment using 3D whole body laser scanning and 2D digital photography in females with a diagnosed eating disorder: preliminary novel findings. *British Journal of Psychology*, 103, 183-202.
- Stice, E. (2001). A prospective test of the dual pathway model of bulimic pathology: mediating effects of dieting and negative affect. *J. Abnorm. Psychol.*, 110, 1–12.
- Stice, E., & Agras, W.S. (1999). Subtyping bulimic women along dietary restraint and negative affect dimensions. *Journal of Consulting and Clinical Psychology*, 67, 4, 460-469.

- Stice, E., Hayward, C., Cameron, R.P., Killen, J.D., & Taylor, C.B. (2000). Body-image and eating disturbances predict onset of depression among female adolescents: a longitudinal study. *Journal of Abnormal Psychology*, 109, 438-44.
- Stice, E., Schupak-Neuberg, E., Shaw, H. E., & Stein, R. I. (1994). Relation of media exposure to eating disorder symptomatology: an examination of mediating mechanisms. *Journal of abnormal psychology*, 103(4), 836.
- Stice, E., & Shaw, H.E. (2002). Role of body dissatisfaction in the onset and maintenance of eating pathology: a synthesis of research findings. *Journal of Psychosomatic Research*, 53, 985-993.
- Stonehill, E., & Crisp, A. H. (1977). Psychoneurotic characteristics of patients with anorexia nervosa before and after treatment and at follow-up 4–7 years later. *Journal of Psychosomatic Research*, 21(3), 187-193.
- Stormer, S. M., & Thompson, J. K. (1996). Explanations of body image disturbance: A test of maturational status, negative verbal commentary, social comparison, and sociocultural hypotheses. *International Journal of Eating Disorders*, 19(2), 193-202.

- Striegel-Moore, R.H. (1995). A feminist perspective on the etiology of eating disorders. In: Brownell, K.D., Fairburn, C.G., eds. *Eating Disorder and Obesity. A Comprehensive Handbook*. New York: The Guilford Press, 224–229.
- Striegel-Moore, R.H., Franko, D.L., Thompson, D., Barton, B., Schreiber, G.B., & Daniels, S.R. (2004). Changes in weight and body image over time in women with eating disorders. *International Journal of Eating Disorders*, 36, 315-327.
- Strober, M., Freeman, R., & Morrell, W. (1997). The long-term course of severe anorexia nervosa in adolescents: Survival analysis of recovery, relapse, and outcome predictors over 10-15 years in a prospective study. *International Journal of Eating Disorders*, 22, 4, 339-360.
- Strober, M., Freeman, R., Lampert, C., Diamond, J., & Kaye, W. (2000). Controlled family study of anorexia nervosa and bulimia nervosa: Evidence of shared liability and transmission of partial syndromes. *American Journal of Psychiatry*, 157, 3, 393-401.
- Stunkard, A.J., Sorensen, T., & Schulsinger, F. (1982). Use of the Danish Adoption Register for the study of obesity and thinness. *Research Publications - Association for Research in Nervous & Mental Disease*, 60, 115–120.

- Suchan, B., Busch, M., Schulte, D., Gronemeyer, D., Herpertz, S., & Vocks, S. (2010). Reduction of gray matter density in the extrastriate body area in women with anorexia nervosa. *Behavioural Brain Research*, 206, 63–67.
- Swanson, S. A., Horton, N. J., Crosby, R. D., Micali, N., Sonnevile, K.R., Eddy, K., & Field, A.E. (2014). A latent class analysis to empirically describe eating disorders through developmental stages. *International Journal of Eating Disorders*, 47, 7, 762-772.
- Szymanski, M. L., & Cash, T. F. (1995). Body-image disturbances and self-discrepancy theory: Expansion of the Body-Image Ideals Questionnaire. *Journal of Social and Clinical Psychology*, 14, 134–146.
- Tanner, C.M., Ottman, R., Goldman, S.M., Ellenberg, J., Chan, P., Mayeux, R., & Langston, J.W (1999). Parkinson disease in twins: an etiologic study. *The Journal of the American Medical Association*, 281(4), 341–346.
- Tapajóz, F., de Sampaio, P., Soneira, S., Aulicino, A., Harris, P., & Allegri, RF. (2015). Emotional reactivity to social stimuli in patients with eating disorders. *Psych Res*, 229, 887-894.

Taylor, R.W., Keil, D., Gold, E.J., Williams, S.M., & Goulding, A. (1998). Body mass index, waist girth, and waist-to-hip ratio as indexes of total and regional adiposity in women: evaluation using receiver operating characteristic curves. *American Journal of Clinical Nutrition*, 67, 1, 44-49.

Thompson, J. K., Coover, M. D., Richards, K. J., Johnson, S., & Cattarin, J. (1995). Development of body image, eating disturbance, and general psychological functioning in female adolescents: Covariance structure modeling and longitudinal investigations. *International Journal of Eating Disorders*, 18(3), 221-236.

Thompson, J. K., & Heinberg, L. J. (1999). The media's influence on body image disturbance and eating disorders: We've reviled them, now can we rehabilitate them?. *Journal of social issues*, 55(2), 339-353.

Thompson, J.K., Heinberg, L.J., Altabe, M., & Tantleff-Dunn, S. (1999). *Exacting beauty: theory, assessment, and treatment of body image disturbance*. Washington (DC): American Psychological Association.

Thompson, D.L., Thompson, W.R., Prestridge, T.J., Bailey, J.G., Bean, M.H., Brown, S.P., & McDaniel, J.B. (1991). Effects of hydration and dehydration on body composition analysis: a comparative study of bioelectric impedance

analysis and hydrodensitometry. *Journal of Sports Medicine and Physical Fitness*, 31, 4, 565-570.

Thompson, R., & Zuroff, D. C. (1999). Development of self-criticism in adolescent girls: Roles of maternal dissatisfaction, maternal coldness, and insecure attachment. *Journal of Youth and Adolescence*, 28(2), 197-210.

Tiggemann, M. (2003). Media exposure, body dissatisfaction and disordered eating: Television and magazines are not the same!. *European Eating Disorders Review*, 11(5), 418-430.

Tiggemann, M., & McGill, B. (2004). The role of social comparison in the effect of magazine advertisements on women's mood and body dissatisfaction. *Journal of Social and Clinical Psychology*, 23(1), 23.

Tovée, M. J. (2012). Anthropometry. In: T.F. Cash, Ed. *Encyclopedia of Body Image and Human Appearance*. San Diego: Academic Press.

Tovée, M.J., Benson, P.J., Emery, J.L., Mason, S.M., & Cohen-Tovée, E.M. (2003). Measurement of body size and shape perception in eating-disordered and control observers using body-shape software. *British Journal of Psychology*, 94, 501-516.

- Tovée, M.J., Edmonds, L., & Vuong, Q.C. (2012). Categorical perception of human female physical attractiveness and health. *Evolution and Human Behaviour*, 33, 85-93.
- Tovée, M. J., Emery, J. L., & Cohen-Tovée, E. M. (2000). The estimation of body mass index and physical attractiveness is dependent on the observer's own body mass index. *Proceedings of the Royal Society B: Biological Sciences*, 267, 1987–1997.
- Tovée, M. J., Maisey, D. S., Emery, J. L., & Cornelissen, P. L. (1999). Visual cues to female physical attractiveness. *Proc. R. Soc. Lond. B*, 266, 211–218.
- Tovée, M.J., Swami, V., Furnham, A., & Mangalparsad, R. (2006). Changing perceptions of attractiveness as observers are exposed to a different culture. *Evolution and Human Behaviour* 2006, 27(6), 443-456.
- Traub, A.C., & Orbach, J. (1964) Psychophysical studies of body-image: 1. The adjustable body-distorting mirror. *Archives of General Psychiatry*, 11, 1, 53-&.
- Treasure, J., Claudino, A. M., & Zucker, N. (2010). Eating disorders. *Lancet*, 375,583–593.

- Treasure, J., & Collier, D. (2000). The relationship of the fat controller to eating disorders. *European Eating Disorders Review*, 8, 3, 193-197.
- Treasure, J., Todd, G., Brolly, M., Tiller, J., Nehmed, A., & Denman, F. (1995). A pilot study of a randomised trial of cognitive analytical therapy vs educational behavioural therapy for adult anorexia nervosa. *Behaviour research and therapy*, 33(4), 363-367.
- Truesdale, K., & Stevens, J. (2008). Do the obese know they are obese? *North Carolina Medical Journal*, 69, 188–194.
- Uher, R., Murphy, T., Friederich, H.C., Dalglish, T., Brammer, M.J., Giampietro, V., & Treasure, J. (2005). Functional neuroanatomy of body shape perception in healthy and eating disordered women. *Biological Psychiatry*, 58, 990–997.
- Unger, T.J., Calderon, G.A., Bradley, L.C., Sena-Esteves, M., & Rios, M. (2007). Selective deletion of Bdnf in the ventromedial and dorsomedial hypothalamus of adult mice results in hyperphagic behaviour and obesity. *Journal of Neuroscience*, 27, 14265–14274.

- Urgesi, C., Fornasari, L., Perini, L., Canalaz, F., Cremaschi, S., Faleschini, L., & Brambilla, P. (2012). Visual body perception in anorexia nervosa. *International Journal of Eating Disorders*, 45(4), 501-511.
- van Beers, R.J., Baraduc, P., & Wolpert, D.M. (2002). Role of uncertainty in sensorimotor control. *Philosophical Transactions of the Royal Society B – Biological Sciences*, 357, 1137–1145.
- van den Berg, P., Thompson, J.K., Obremski-Brandon, K., & Covert, M. (2002). The Tripartite Influence model of body image and eating disturbance: A covariance structure modeling investigation testing the mediational role of appearance comparison. *Journal of Psychosomatic Research*, 53, 1007– 1020
- Vervaet, M., van Heeringen, C., & Audenaert, K. (2004). Binge eating disorder and non-purging bulimia: More similar than different? *European Eating Disorders Review*, 12, 1, 27-33.
- Vink, T., Hinney, A., van Elburg, A.A., van Goozen, S.H.M., Sandkuijl, L.A., Sinke, R.J. et al. (2001). Association between an agouti-related protein gene polymorphism and anorexia nervosa. *Molecular Psychiatry*, 6, 325–328.

- Vocks, S., Busch, M., Gronemeyer, D., Schulte, D., Herpertz, S., & Suchan, B. (2010a). Neural correlates of viewing photographs of one's own body and another woman's body in anorexia and bulimia nervosa: an fMRI study. *Journal of Psychological Neuroscience*, 35(3), 163-176.
- Vocks, S., Busch, M., Gronemeyer, D., Schulte, D., Herpertz, S., & Suchan, B. (2010b). Differential neuronal responses to the self and others in the extrastriate body area and the fusiform body area. *Cognitive, Affective and Behavioural Neuroscience*, 10, 422-429.
- Vocks, S., Schulte, D., Busch, M., Gronemeyer, D., Herpertz, S., & Suchan, B. (2011). Changes in neuronal correlates of body image processing by means of cognitive behavioural body image therapy for eating disorders: a randomized controlled fMRI study. *Psychological Medicine*, 41, 1651-1663.
- von Wietersheim, J., Kunzl, F., Hoffmann, H., Glaub, J., Rottler, E., & Traue, H.C. (2012). Selective attention of patients with anorexia nervosa while looking at pictures of their own body and the bodies of others: an exploratory study. *Psychosom Med*, 74, 107-13.
- Voracek, M., & Fisher, M. L. (2002). Shapely centrefolds? Temporal change in body measures: trend analysis. *British Medical Journal*, 325(7378), 1447.

- Vreugdenburg, L., Bryan, J., & Kemps, E. (2003). The effect of self-initiated weight-loss dieting on working memory: the role of preoccupying cognitions. *Appetite, 41*(3), 291-300.
- Wagner, A., Aizenstein, H., Venkatraman, V., Fudge, J., May, J., Mazurkewicz, L., & Kaye, W.H. (2007). Altered reward processing in women recovered from anorexia nervosa. *American Journal of Psychiatry, 164*, 1842-1849.
- Walsh, B. T., & Sysko, R. (2009). Broad Categories for the Diagnosis of Eating Disorders (BCD-ED): An alternative system for classification . *International Journal of Eating Disorders, 42*, 8, 754-764.
- Ward, A., Ramsay, R., & Treasure, J. (2000). Attachment research in eating disorders. *British Journal of Medical Psychology, 73*(1), 35-51.
- Wardle, J., & Watters, R. (2004). Sociocultural influences on attitudes to weight and eating: results of a natural experiment. *International journal of eating disorders, 35*(4), 589-596.
- Warren, C.J., & Cooper, P.J. (1988). Psychological effects of dieting. *British Journal of Clinical Psychology, 27*, 3, 269-270.

Watson, H., & Bulik, C. (2013). Update on the treatment of anorexia nervosa: review of clinical trials, practice guidelines and emerging interventions. *Psychological Medicine* 43, 12, 2477–2500.

Watson, K.K., Werling, D.M., Zucker, N.L., & Platt, M.L. (2010). Altered social reward and attention in anorexia nervosa, *Frontiers in Psychology*, 1, 36.

Watt, R.J., & Andrews, D.P. (1981) APE: Adaptive probit estimation of psychometric functions. *Current Psychology Review*, 1, 205-214.

Watts, K., Naylor, L.H., Davis, E.A., Jones, T.W., Beeson, B., Bettenay, F., & Green, D.J. (2006). Do skinfolds accurately assess changes in body fat in obese children and adolescents? *Medicine and Science in Sports and Exercise*, 38, 3, 439-444.

Wellcome Trust Case Control Consortium (2007). Genome-wide association study of 14,000 cases of seven common diseases and 3,000 shared controls. *Nature*, 447, 661–678.

Wells, A.S., Read, N.W., Laugharne, J.D.E., & Ahluwalia, N.S. (1998). Alterations in mood after changing to a low-fat diet. *British Journal of Nutrition*, 79, 1, 23-30.

- Wells, J.C., Treleaven, P., & Cole, T.J. (2007). BMI compared with 3-dimensional body shape: the UK national sizing survey. *The American Journal of Clinical Nutrition*, 85, 419-425.
- Wells, J.C.K., Cole, T.J., Bruner, D., & Treleaven, P. (2008). Body shape in American and British adults: Between-country and inter-ethnic comparisons. *International Journal of Obesity*, 32, 152–159.
- Weschler, D. (1981). Weschler Adult Intelligence Scale – Revised. The Psychological Corporation, Harcourt Brace Janovich.
- Westen, D., & Harnden-Fischer, J. (2001). Personality profiles in eating disorders: Rethinking the distinction between axis I and axis II. *American Journal of Psychiatry*, 158, 4, 547-562.
- Wetmore, C., & Modkdad, A.H. (2012). In denial: misperceptions of weight change among adults in the United States. *Preventative Medicine*, 56, 93–100.
- Widiger, T.A., & Samuel, D.B. (2005). Diagnostic categories or dimensions? A question for the diagnostic and statistical manual of mental disorders-fifth edition. *Journal of Abnormal Psychology*, 114, 4, 494-504.

- Wildes, J. E., & Marcus, M. D. (2013). Alternative methods of classifying eating disorders: models incorporating comorbid psychopathology and associated features. *Clinical psychology review*, 33(3), 383-394.
- Williamson, D.A., Muller, S.L., Reas, D.L., & Thaw, J.M. (1999). Cognitive bias in eating disorders: implications for theory and treatment. *Behaviour Modification*, 23, 556–577.
- Windauer, U., Lennerts, W., Talbot, P., Touyz, S. W., & Beumont, P. J. (1993). How well are 'cured' anorexia nervosa patients? An investigation of 16 weight-recovered anorexic patients. *The British Journal of Psychiatry*, 163(2), 195-200.
- Winkler, C., & Rhodes, G. (2005). Perceptual adaptation affects attractiveness of female bodies. *British Journal of Psychology*, 96, 141–154.
- Wonderlich, S. A., Joiner, T. E., Keel, P. K., Williamson, D.A., & Crosby, R.D. (2007). Eating disorder diagnoses - Empirical approaches to classification. *American Psychologist*, 62, 3, 167-180.
- Wonderlich, S. A., Lilenfeld, L. R., Riso, L. P., Engel, S., & Mitchell, J. E. (2005). Personality and anorexia nervosa. *International Journal of Eating Disorders*, 37(S1), S68-S71.

Woods, S.C., & D'Alessio, D.A. (2008). Central control of body weight and appetite. *Journal of Clinical Endocrinology*, 93(11 Suppl. 1), S37-S50.

Yager, Z., & O'Dea, J.A. (2011). A controlled intervention to promote a healthy body image, reduce eating disorder risk and prevent excessive exercise among trainee health education and physical education teachers. *Health Education Research*, 25, 5, 841-852.

Yarbus, A.L. (1967). *Eye Movements and Vision*. New York: Plenum Press, 1967.

Yates, A., Edman, J., & Aruguete, M. (2004). Ethnic differences in BMI and body/self-dissatisfaction among Whites, Asian subgroups, Pacific Islanders, and African-Americans. *Journal of Adolescent Health*, 34(4), 300-307.

Zipfel, S., Wild, B., Groß, G., Friederich, H. C., Teufel, M., Schellberg, D., & Burgmer, M. (2014). Focal psychodynamic therapy, cognitive behaviour therapy, and optimised treatment as usual in outpatients with anorexia nervosa (ANTOP study): randomised controlled trial. *The Lancet*, 383(9912), 127-137.